4-1-1965

Volume 08, issue 2

Canadian Medical Association

HISTORY OF CANADIAN SURGERY

IRVING HEWARD CAMERON (1855-1933)
Professor of Surgery, University of Toronto, 1897-1920

C. W. HARRIS, M.D., F.R.C.S.[C],* *Toronto, Ont.*

CAPAX, PERSPICAX, SAGAX ET EFFICAX.
—*John Brown*

THEY BE THE BEST CHIRURGEONS WHO, BEING LEARNED, INCLINE TO THE TRADITIONS OF EXPERIENCE, OR, WHO BEING EMPIRICS, INCLINE TO THE METHODS OF LEARNING.—*Bacon*

HE WAS known to us all as Mr. Cameron. He was quite insistent on the title. This was no pose in mimicry of Old Country surgeons but was because he was a stickler for the proper use of words. To him, "doctor" meant teacher and should not be wasted on every medical practitioner. He himself might well have been called "doctor" for he taught and wished each of his students to become "a philosophic physician (a natural philosopher), a priest of nature and not a rule-of-thumb practitioner of the healing art, an artisan or handicraftsman".[3] John Brown and Francis Bacon, often quoted by him, had specified the ingredients of the good surgeon and these qualities were his.

The Camerons were an old and illustrious family of Fassiefern in Scotland. The grandfather, John McAlpine Cameron, had come from Liverpool to Upper Canada in 1819 and had gone with his two sons to Dundas to engage in mercantile pursuits. These two sons studied law and practised in Hamilton. A third son, Mathew Crooks, was born in Canada and attended Upper Canada College in Toronto. A hunting accident caused the amputation of a leg resulting in the necessary choice of a sedentary life. He articled with Gamble and Boulton and was called to the bar in 1849. He tried politics, unsuccessfully at first because of his staid demeanour, his high sense of honour and independence and his reluctance for enthusiastic handshaking. He did have a lively and sociable disposition and the magnanimity of a Scottish chief, all attributes that were to appear again in his son, Irving Heward Cameron. Early in his career at the bar he defended a slave who had escaped and crossed the border and whose owner wished to recover him. His success in this case set a precedent that was quite unwelcome to slave-owners and also caused some friction between the governments concerned. Mathew Crooks Cameron, a Tory of Tories, did achieve some political success later; he was among those who prepared the way for Confederation, and afterwards sat in the Provincial House and was Provincial Secretary. His success and experience at the bar qualified him for a judgeship, and, as Sir Mathew Crooks Cameron, K.C.M.G., he presided as Chief Justice of the Court of Common Pleas. One of his

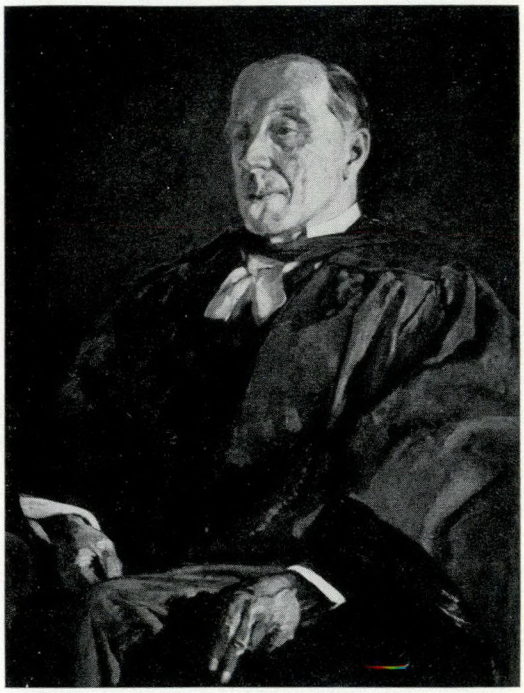


Fig. 1.—Irving H. Cameron, from the portrait by Fred Varley in the Faculty of Medicine, Toronto.

*Suite 342, Professional Suites, Toronto Western Hospital, Toronto 2B.

great interests had been medical jurisprudence.[1]

Irving Heward Cameron was the son of this man and of Jessie Ross Wedd of Maidstone, Kent. He attended grammar school and then at the age of 12 was sent to Upper Canada College like his father. His six years there were uneventful; his name did not appear on a scholarship list, nor was he ever head boy, but there he gained his first knowledge of Latin and Greek and a love of literature and letters, interests that abided with him throughout his life and which were abetted by an unusual memory. Beyond this schooling it is not on record that he ever attended University to receive formal training in the *literae humaniores* but he obtained a mastery of them sufficient for him to be at ease among scholars; they served as an ever-ready source of quotations which in Greek, Latin, English or occasionally in French coloured his sayings and his writings. His cultural attainments spoke well for both his teachers and his home life. It was these quotations that have made him so well remembered by his students and although they were rarely understood they pointed the way to scholarship and culture, and hopefully, to emulation.

His first choice for a career had been law, but after a few months his interest turned to medicine. He entered the Toronto School of Medicine in 1871 and three years later received his M.B. from the University of Toronto. At the school, H. H. Wright was Professor of Medicine. Wright had attended the first Rolph School and had gone into exile with Rolph in 1837. Political differences were no bar to romance, for the son of the arch-Tory, Cameron, married the daughter of the Radical, Wright.

He then spent some time in England and Scotland and perhaps there enlarged his classical knowledge. He has left on record his admiration for and debt to Burdon Sanderson, Hughlings Jackson, James Paget and Richard Quain. There he adopted Listerism, recently promulgated, and he remained an exponent of Lister's methods long after others had been converted to asepticism. Osler had made the grand tour in Europe just two years before. On Osler's death in 1919, Mr. Cameron, in a tribute to him, was able to speak of the men whom Osler and he had come in contact with and had been influenced by.

By 1880 he was practising in Toronto. Like any other doctor of the time any illness was grist to his mill. The journals of the time, recording the meetings of the Toronto Clinical Society, show him to have been a regular attendant and contributor. He seemed to have had a particular interest in skin diseases.

Early in his career he was elected to the Senate of the University of Toronto where he was to serve for many years and early became attached as a teacher to his old school of medicine.

The problems that were to present themselves were the organization of the Faculty of Medicine, the rebuilding of the Toronto General Hospital on a new site and the moulding of university teaching in medicine to produce a broad standard of education. After these came the establishment of a proper surgical department in the hospital. To each in turn he brought a cultivated mind and personality that supported his arguments against sometimes strenuous opposition. He said he learned his surgery from W. T. Aikins, Professor of Surgery at Toronto, whom he revered and who was one of the strongest advocates of the new Faculty. With its formation in 1887 he became Professor of the Principles of Surgery and of Surgical Pathology and five years later he succeeded Aikins in the chair of Surgery and Clinical Surgery.

Throughout the years the University's service claimed Mr. Cameron's unswerving devotion, often at great personal sacrifice. Neither consultation nor operation was allowed to interfere with a meeting of a committee of the Senate; such personal matters had to be postponed.

The rebuilding of the hospital was not the accomplishment of a moment. A decided impetus to its construction was the formation of the new Faculty and the consequent demand for proper facilities for student instruction, but to persuade City and Provincial Governments of their responsibility, to gain their financial sup-

port, and to enlist the growing number of wealthy, prominent and public-spirited citizens, who ultimately erected the institution, was a matter of patient persuasion and time. In this Mr. Cameron played his full part. Here is a letter which he wrote in 1906 to the Honourable James P. Whitney, Premier of Ontario at that time.[2]

MR. IRVING H. CAMERON
S. E. SHERBOURNE & GERRARD STS.
11 A.M.—1 P.M. 7 P.M.—9 P.M.
TELEPHONE 1317

4 April, 1906.

Dear Mr. Premier:

I don't know whether the Hospital Bill has been reported yet or not but I suppose you will likely speak upon the 3rd Reading anyway and if I might venture to impose upon your already overburdened attention I would like to present two or three points for your consideration. Your Government is not responsible for State Aid to Medical Education and wholly proper and defensible—nay commendable—though it be I do not know whether you approve it or not. But, no matter! This is an existing condition which you found and no doubt, will make the best of it. The Government through the Provincial University provides Laboratory instruction and facilities for Medical Students in the first two years of this Course. The chief laboratory for the final two years are the Hospital Wards and this circumstance alone justifies the Government and University and its Medical Faculty in contributing Half a Million Dollars between them to the projected New Hospital in Toronto.

Now I suppose it is apparent that we (the parties above named of the first part) shall be making a very bad bargain and doing a very unbusinesslike thing if it is not specified, understood and agreed between the parties that the New Hospital Trustees (the parties of the second part) will provide such clinical facilities and make such Staff Appointments as required from time to time by the exigencies of the University. Unless these two considerations are made absolutely certain the University cannot afford to go into the scheme but should on the other hand insist upon having a smaller independent University Hospital. Again, all the teaching done in this Hospital should be done by persons authorized by the Gov't to teach, appointed and controlled by the Lieut.-Governor in Council and by none other. Otherwise there is no means of regulating the character of the teaching.

As things now stand, out of 375 beds 220 are available for clinical teaching, a proportion in a public Hospital ridiculously small.

The name, Toronto General Hospital, is not, like the University of Toronto, associated with a brilliant past and I would suggest that it might be advantageously changed to The Toronto Royal Infirmary and University Hospital. The Royal consent could doubtlessly be easily obtained and perhaps associated with the projected visit of the Prince of Wales. And it would add to the New Institution the Prestige of the Royal Patronage and of a closer association with the university. You may perhaps think that I overrate the latter, but, if so I must plead in extenuation that I adhere to the old-fashioned notion of "a seat of sound learning" and not an advanced technical school.

With due apologies,
I. H. CAMERON

Other hospitals received his help. From its inception he was surgeon to the Hospital for Sick Children where a most valuable contribution was the arrangement for adequate facilities for clinical instruction. Here his friendship with John Ross Robertson, the Hospital's founder, gained him a ready support. St. John's Hospital for Women received his help in its organization and there he did much of his private surgery. The Sisters of St. John readily acknowledged his help and support and his part in the establishment of the high standard which always marked that hospital. St. Michael's Hospital too at one time numbered him among its staff.

At the time of his first teaching appointment, surgeons here and elsewhere did not limit themselves to the practice of surgery. Surgeons were those physicians with a flair for handicraft and perhaps the opportunity of an apprenticeship. By the time he was heading his department, Halstead of Johns Hopkins had shown that proper training could be had and able, young and trained surgeons were becoming available. The Old Country-orientated Professor found Canadians who had spent considerable time in New York and other American hospitals; C. L. Starr, W. E. Gallie and

Norman Shenstone were added to his department from American sources and have made their school's reputation. George Peters, the only Toronto surgeon with a European reputation at the time, was English-trained and was appointed by Mr. Cameron. Alexander Primrose, one of his best teachers, came from Edinburgh by way of the Department of Anatomy. H. A. Bruce, the future Lieutenant-Governor of the Province, was another new member of the staff. Dr. Bruce has pointed out that his advent was not altogether welcome because his appointment followed other than hospital or university recommendation, but he became a distinct addition to the staff. Others on his staff were F. N. G. Starr, George Bingham, John Malloch, C. B. Shuttleworth and George Wilson.

Dr. Shenstone was devoted to his chief. He has this to say of their association: "Mr. Cameron was by present standards not a great surgeon, but the rather simple operations that he undertook during the years of our association were clearly and carefully performed and I can recall no infection in a clean case. It was stated in a recent biography that he always had one of his staff to assist him in his operations. This is quite true but the fact of the matter is that nearly all surgeons of that time were accustomed to having a staff member as their assistant. The reason for this was that the interns were, in the early 1900's, appointed for only 6 to 12 months and could not be given enough training to be satisfactory. The operating-room set-up would usually place the intern at a table from which he handed the instruments to the surgeon as they were required. The intern therefore had very little experience as an assistant and would have been greatly in the way of the operator.

"Mr. Cameron was in 1909 still a firm adherent of the antiseptic surgery of the preceding 30 years. He could not be induced to wear gloves, giving as his reason that with them he lost the sensitivity of his fingers. He prepared his hands and forearms by washing them with powdered mustard and then soaking them in bichloride of mercury or carbolic acid solution. He used sea sponges rather than the gauze sponges which were standard at that date. They were kept in a carbolic solution between operations and were washed by the nurse when soiled during the operation. They would subsequently, after thorough cleansing, be kept in a carbolic solution until the next operation. Keith's dressing was his favourite for his operative wounds."

He used very few instruments in an operation. His skin sutures were horsehair from the mane of a black stallion. If he kept his operations simple and avoided the daring, it was in part because of his belief that the surgeon should always put himself in the position of the patient and think of how he would like to be treated under such a circumstance, and also because at the time of his active career, surgery had not advanced to the stage that became familiar to his juniors. "Never operate", he said, "just to see what is inside." Not for him the exploratory laparotomy! He had the presence of mind and ability to think out the best solution of a difficult problem on the spur of the moment. There was one occasion when his quiet advice to another surgeon, rattled by an unsuspected emergency, saved the patient's life. He had chanced to come into the operating-room a minute before.

As a teacher it was to the worthy few that he appealed. To these he taught principles and the importance of principles, aiming to train his students to think their problems out by themselves and to teach them the importance of the correct use of words. His love of words and ideas would carry him away from his desired topic. The greater part of his lecture was often devoted to the events of which the day was the anniversary or to the meaning and derivation of an unusual word. His students tried to look as if they understood his Latin and Greek quotations. One remembered clinic was about bee-stings and what Ovid and Horace thought of them, and another was on intestinal obstruction which closed with the chuckled remark, "*qui crepitat vivat*". If it were an outpouring of facts his students wanted, they needed to go elsewhere.

The story is told that the late Mr. Timothy Eaton had fallen and injured his

hip. Mr. Cameron was called in and during the course of his examination caused the patient pain; the latter understandably used his uninjured limb to violently push the doctor away. Mr. Cameron went to the bottom of the room, picked up his hat and gloves, bowed and left the room. Dr. Gideon Silverthorn then attended the patient.

Outside of the University he engaged in numerous professional and public activities. He was one of the founders of the *Canadian Journal of Medical Sciences.* He was instrumental in the establishment of the *University of Toronto Monthly* and for years served on its editorial board, contributing many articles to it.

He was in England when the First Great War broke out and promptly joined the Royal Army Medical Corps. Later he was transferred to the C.A.M.C. and established the surgical department of the Ontario Hospital for wounded soldiers at Orpington, Kent. With the establishment of the Canadian Red Cross Hospital at Taplow he was able to render a similar service. After the war he was appointed to the Board of Consultants for the 70 military hospitals in Canada. In spite of this added responsibility, he continued his teaching and the direction of the Surgical Department at the Toronto General Hospital until his retirement in 1920.

Because of his own attainments and in tribute to his school, numerous honours came his way. The Royal College of Surgeons of England had gained the power to elect Honorary Fellows up to a maximum number of 50 under a Supplementary Charter dated December 22, 1899. During 1900 the Council of the College therefore drew up a list of the first recipients of the Honorary Fellowship. The Prince of Wales, the Earl of Rosebery and the Marquess of Salisbury were elected in the first half of the year and on July 25, 1900, the Council formally elected 35 other Honorary Fellows from a number of countries. This coincided with the celebration of the College's Centenary and all of the Honorary Fellows, with the exception of the Prince of Wales, attended the Centenary Meeting on July 26 "in the theatre in Burlington Gardens lately used by the University of London" to receive their diplomas. One of the speeches in reply to the address of welcome by the President of the College, Sir William MacCormac, was by Dr. T. G. Roddick, on behalf of the three Honorary Fellows from Canada, himself, Sir William Hingston and Professor Cameron. The Royal Colleges of Edinburgh and Dublin similarly honoured him, and Edinburgh added an LL.D. He was a Charter Fellow of the American College of Surgeons and a Fellow of the Royal College of Surgeons of Canada.

The International Society of Surgeons claimed him as a member, as did the British Association for the Advancement of Science and the American Academy of Political and Social Science. In 1898 he presided at the meeting of the Canadian Medical Association in Quebec and the next year was president of the Toronto Branch of the British Medical Association.

He was a man of all too rare personal qualities with, above all, the gift of equanimity. He had great personal charm and was fastidious in everything—in his use of language, in his dress, the turnout of his dog-cart, even in the jaunty angle with which he carried his whip. He was a man of unfailing courtesy and liberal hospitality, with an impressive dignity of presence and an austere expression ready to light up when his interests were touched, whether it was by medicine, by horses or by the history of the Cameron clan. Although he made few contributions to medical literature he contributed many essays on non-medical subjects and found time to edit in turn the *Canadian Journal of Medicine and Surgery,* the *Canadian Journal of Medical Sciences* and the *University of Toronto Monthly.* All his writings showed the same thought, care and apt quotations that appeared in his spoken word.

For his epitaph one might use his own words written more than 60 years ago.[3]

"I am fain to confess a weakness for the old *literae humaniores* in the education of a physician, as of every other gentleman, whose experience will be concerned with the gentler and nobler, and less strenuous and more

human side of life . . . . . as to prove a never failing cruse of oil to the lamp of science which has been burning with ever increasing brilliancy for fifty years in 'this gorgeous temple of infidelity', which mingled with the darkness and was not comprehended of it, but which sufficed to illuminate the smithy in which was forged a yearly 'link betwixt us and the crowning race of those who eye to eye shall look on knowledge, and in whose hand is Nature like an open book' a book wherein 'the anointed eye may trace'—not 'a dead soul's epitaph', but the unending upward progress of the human race."

The writer wishes to thank Miss Mary Fawkes and Mr. George Fawkes of Brantford and Mrs. Gordon Murray of Toronto, members of the Cameron Clan, for their help. Liberal use has been made of the published appreciation[4] of the late Professor Thomas McCrae.

REFERENCES

1. READ, D. B.: Lives of the judges of Upper Canada and Ontario from 1791 to the present time, Rowsell and Hutchinson, Toronto, 1888.
2. CAMERON, I. H.: Unpublished letter to Honourable J. P. Whitney, Premier of Ontario, Ontario Archives, Record Group 2, University of Toronto Series, 4 April, 1906.
3. CAMERON, I. H.: A university training as a preparation for the medical profession, *University of Toronto Monthly*, **2:** 92, 1902.
4. MCCRAE, T.: An appreciation, *Canad. Med. Ass. J.*, **30:** 225, 1934.

MANUAL OF SURGICAL ANATOMY. Sir John Bruce, R. Walmsley and J. A. Ross. 562 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1964. $16.25.

Several generations of surgeons remember with respect and affection the book of the same title which they almost always referred to as "Beesly and Johnston". For them the appearance of this new book marks the end of an era, because it is an outgrowth of Beesly and Johnston's fifth edition. Aside from the euphony of their names, little actually remains of the contributions of the original authors. For 30 years, the responsibility for revisions has been in the hands of Sir John Bruce and Professor Robert Walmsley. With the addition of a third author and a drastic revision of the text, the book has now entered a new era with a "first" edition.

Because of this background, "Manual of Surgical Anatomy" should have a professional gloss and competence that one does not expect to find with new books in this field. In any event, the happy constellation of a distinguished British anatomist (Walmsley) and two outstanding surgeons could be expected to produce a first-rate book. The reader is not disappointed.

In an abbreviated form, the manual presents the anatomical basis of surgical practice, taking advantage here and there of classical operations to present detailed anatomical descriptions. Thus, two obvious targets of this volume are the senior clinical student and the surgical trainee. Both will be rewarded if they become intimately familiar with it.

As might be expected, the longest chapter deals with the abdominal cavity. Extensive coverage is also given to the head and neck, to the organs of special sense and to the limbs. The chapter on the vertebral column and spinal cord is noteworthy; in fact, writers of standard textbooks of anatomy will be wise to be guided by its approach.

There is much to praise in this book, yet it is not free of fault. The only important error is one of judgment, and here the fault may lie with the present reviewer. In his view, the authors have not taken sufficient advantage of illustrations and have relied too often on words. Admitting that those words are usually well chosen, the reviewer believes that in many areas of anatomical description they are barely sufficient. A larger number of well-prepared special illustrations would have turned this fine book into a superb one. The almost assured success of this edition should encourage the authors to embark on the preparation of a larger number of drawings for the next edition. In this way, they can turn this book into a classic in their own lifetime.

SEPTIC SHOCK. Experimental and Clinical Studies. Hiroshi Hayasaka and John M. Howard. 86 pp. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1964. $6.00.

This little monograph reviews the experimental work that has been performed with endotoxins and exotoxins and also some of the hypotensive conditions associated with severe infections with both Gram-positive and Gram-negative organisms. There is a fairly extensive bibliography.

ORIGINAL ARTICLES

PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY*

MAURICE PARENT, M.D., F.R.C.S.[C], F.A.C.S.,† MAURICE DUFRESNE, M.D.‡ and GUY DUTRON, M.D.,§ *Montreal, Que.*

THE technique, percutaneous transhepatic cholangiography, consists of opacifying the bile ducts by intrahepatic injection of a contrast medium directly through the abdominal wall by means of a trocar.

Opacification of the bile ducts by the percutaneous technique was performed for the first time by Burckhardt and Müller in 1921;[22] at that time the injection was made into the gallbladder. It is evident that this requires an accuracy of localization difficult to achieve and involves a definite risk; this is present also in the transcutaneous hepatic technique but to a lesser degree provided that certain precautions are taken. In 1937, Huard and Do-Xuan-Hop[7] used direct transhepatic injection, and in 1942, Royer, Solari and Lanari[21] opacified the bile ducts by percutaneous injection into the gallbladder, operating under peritoneoscopic control. It should be noted that in the first[22] and third methods[14, 21] if adequate delineation of the biliary tree is to be obtained, the cystic duct must be patent, which is commonly not the case.

In 1952, Carter and Saypol[6] shed new light on this technique and brought it to the fore once more. From then on, the essential factors were studied in several medical centres, both in Europe and America, and although publications on the subject were few, they aroused increasing interest. In 1960, Prioton[2] at Montpellier described a new very promising method, using a posterior approach. Numerous variations of his technique have since been described in the literature, but all are based on the same fundamental principles.

*Departments of Surgery and Radiology, Notre Dame Hospital, Montreal.

Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Quebec, January 1964.

†Surgeon, Notre Dame Hospital.

‡Radiologist, Notre Dame Hospital.

§Resident Surgeon, Notre Dame Hospital. Permanent address: 13, rue de Tournai, Ghlin, Belgium.

RÉSUMÉ OF AUTHORS' CLINICAL EXPERIENCE WITH PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY

The present paper is intended to provide an introduction to this subject and is a preliminary to a much more extensive clinical study at present under way at the Notre Dame Hospital, Montreal.

TABLE I.—PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY

	Performed at Notre Dame Hospital up to January 15, 1965		*Carried out by senior author (M.P.)*	
No. of patients	30		17	
Positive	20		11	
Negative	8			
Hepatitis*		4		4
Stenosis of common duct		1		—
Cirrhosis		1		—
No indication		1		—
Unsuccessful		1		—
*Normal***	2		2	

*Proved at operation.
**Normal ducts visualized in 2 cases of hepatitis.

This examination has been carried out on 30 patients at the time of writing, the results of which are presented in Table I. We wish to describe our technique and report on some of the more characteristic cases. We intend in the near future to give more details concerning the experience derived from the 30 cases and to discuss, apart from the bacteriological and cytological findings, those patients upon whom no operations were performed.

TECHNIQUE

At the Notre Dame Hospital, the anterolateral approach is used in which the premedicated patient is placed in the *dorsal decubitus* position on the radiocinematographic table (Figs. 1 and 2). The right hypochondrium, right flank and mesogastrium are prepared with coloured solution

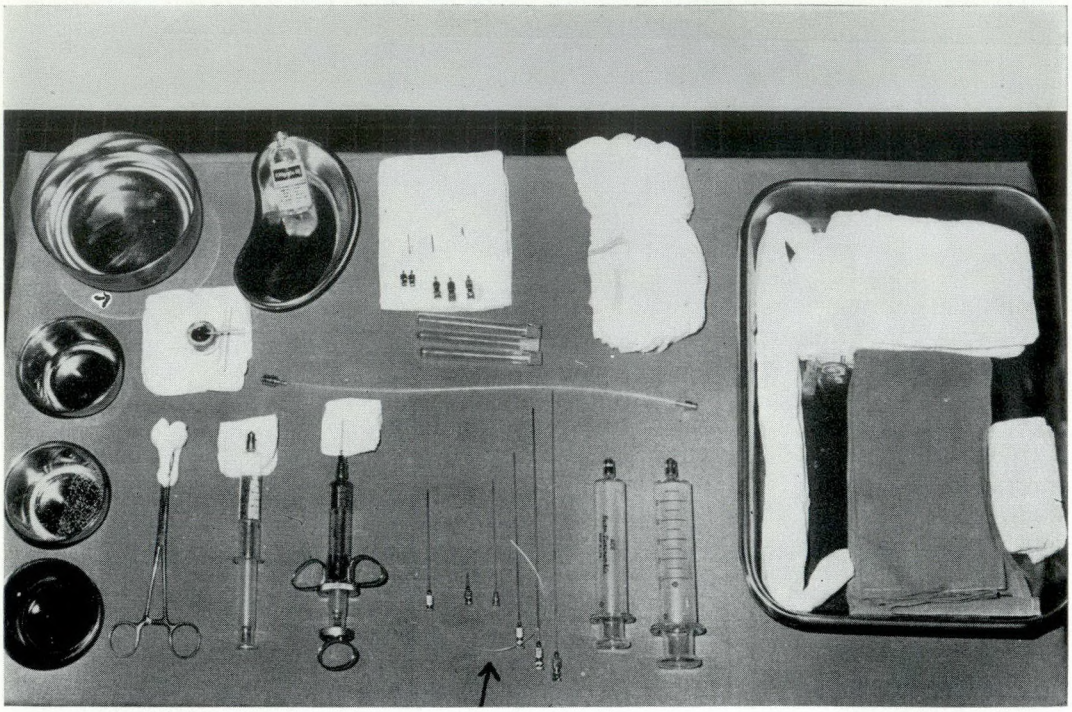


Fig. 1.—Instruments necessary to perform percutaneous transhepatic cholangiography. Note small polythene drainage tube.

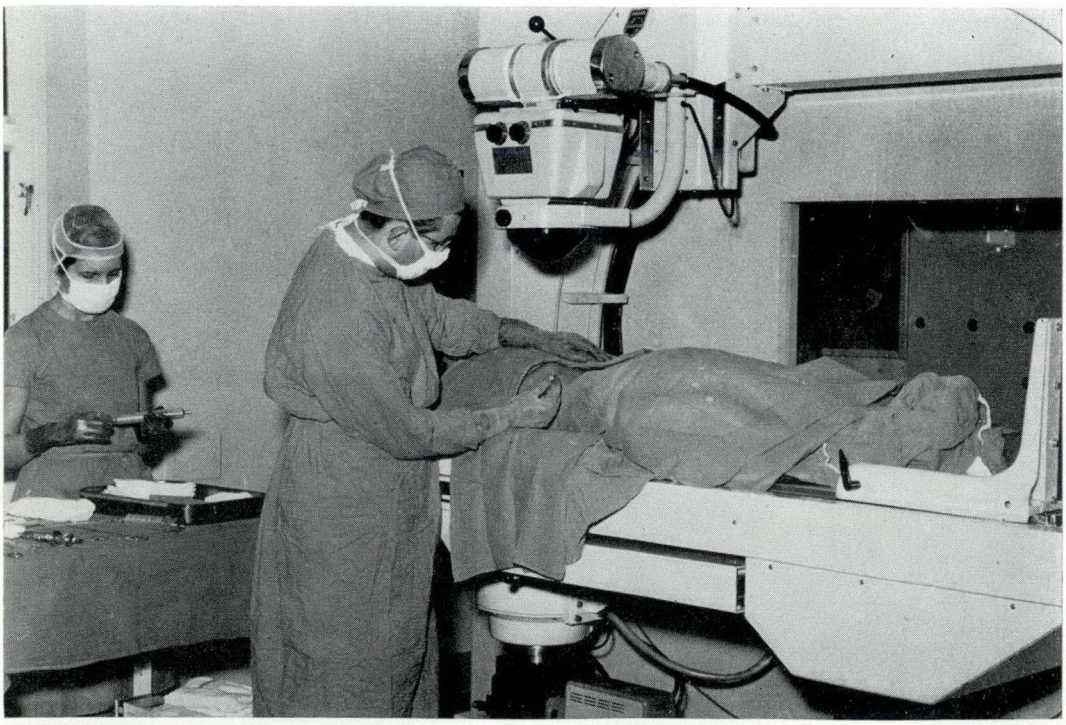


Fig. 2.—This picture shows x-ray equipment needed and stresses the importance of aseptic technique.

of benzalkonium (Zephyran). Sterile drapes are applied. The gloved operator administers local anesthesia using 2% procaine (Zylocain) with epinephrine. The site of injection is at the point of intersection of the anterior axillary line and the upper edge of the tenth or eleventh ribs; at this point the various planes are infiltrated with the local anesthetic up to the parietal peritoneum (Fig. 3). At the above-mentioned site, a 17-gauge trocar is plunged inward, upward and in a slightly anteroposterior direction. The trocar with its stylet is plunged *in the direction of the hilum hepatis*. The trocar is then withdrawn millimetre by millimetre, rotating it about its axis if necessary. At the same time the radiologist checks the position of the trocar on the television screen (Fig. 4). When bile begins to issue forth, the syringe containing 50 c.c. of the contrast medium (Renografin 60%) is attached to the trocar and the contents are injected slowly while the radiologist follows the visualization of the biliary tract on his TV screen. At this time filming is started on demand and standard films are also taken. When the examination is finished, before withdrawing the trocar one should make certain that bile is still issuing; a 30 cm.-long polythene tube is then introduced through the lumen of the trocar and is left in place in the punctured bile duct while the trocar is immediately withdrawn. A sterile dressing is applied and the polythene tube is held in place with adhesive plaster.

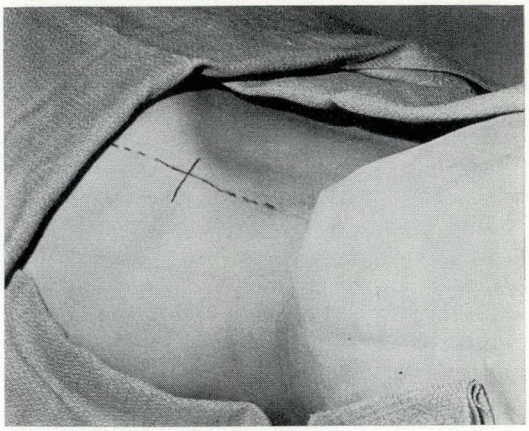


Fig. 3.—Puncture site is at the intersection of the anterior axillary line and the upper edge of the eleventh rib on the right side.

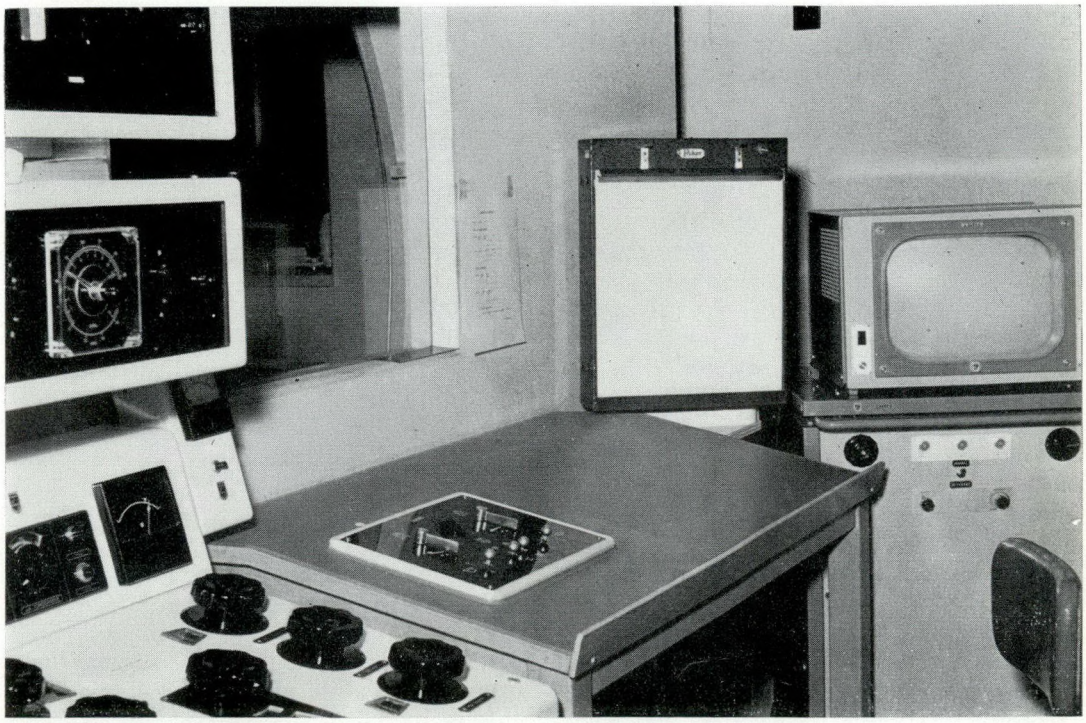


Fig. 4.—This picture shows the equipment that permits the radiologist to film the whole procedure and move the patient as he wishes.

Dangers and Complications

The most frequently reported complications are choleperitoneum (3½% according to a U.S. report[1]) and hemoperitoneum. Either of these complications necessitates a laparotomy within a few hours after the examination. Percutaneous transhepatic cholangiography, when performed *under good conditions* and with the precautions mentioned later is, however, almost without danger. Some rarer complications include pneumothorax and Gram-negative septicemia, which occur chiefly in patients with cholelithiasis and mild stenosis of the common duct and respond quite well to conventional therapy. Subcapsular injection, if observed in time by the radiologist, is of no importance; it will give rise to a slight pain which radiates to the left shoulder, but it is associated with no other complication.

One patient in this series developed choleperitoneum following this examination, and required operation in the ensuing hours; this patient was agitated and had pulled out his polythene tube during the night. In eight other patients who subsequently underwent laparotomy, no blood or bile was found in the peritoneum. No intolerance to the contrast medium has been reported and no complications associated with the injection of the medium occurred in the patients in this series who had hepatocellular jaundice. The technique is not free from danger; nevertheless, experience has shown that in a well-prepared patient the risk is minor if the proper technique is used. The value of the information obtained frequently justifies the use of this procedure.

The following precautions should be observed in order to avoid possible dangers and complications. The examination should be carried out *in a hospital* where an emergency laparotomy can be performed at any time. The patient should be premedicated and the procedure should be explained to him in terms appropriate to his understanding. The *prothrombin level should be normal* (although this examination was carried out on a patient described in the U.S. literature in whom the prothrombin level was only 20% and there were no untoward effects[1]). The patient should be observed for 24 to 48 hours following the procedure for signs of hemorrhage or peritonitis. Some authors recommend a chest radiograph the day after percutaneous transhepatic cholangiography to detect a possible pneumothorax. We have never had pneumothorax after this procedure and we do not routinely perform a radiograph of the chest, although we think it should be done, particularly by those who are not experienced in this technique. We consider that it is important to insert a *polythene tube* in the track of the trocar to drain the bile immediately after the procedure. After 36 to 48 hours, the end of the tube is sealed with heat, and after 72 hours it is removed if nothing untoward has occurred. The only case of choleperitoneum in the present series of cases occurred in the patient who had *pulled out* the polythene tube during the night following the procedure. To prevent accidents this examination should be carried out by a well-trained surgical and radiological team working in *close collaboration* with a competent nursing staff.

Indications

This examination is in no way intended to repace the usual methods of investigating the biliary tract, but is carried out to obtain supplementary information or, in very specific cases, to gain further knowledge when standard procedures have not yielded valid results.

The major indications for percutaneous transhepatic cholangiography are listed in Table II.

It should again be emphasized that this examination should be carried out *only* when the other conventional methods fail to furnish the required information. Percutaneous transhepatic cholangiography can then help to clarify an obscure diagnosis, permitting the selection of the best course of action and at times greatly shortening a tedious investigation on the operating-table. The course to be followed in difficult cases can then be calmly discussed *before operation*, instead of debating when all is over what should perhaps have been done. The quality of the films obtained by this technique is always superior to those obtained of the biliary tree by other meth-

TABLE II.—Indications for Percutaneous Transhepatic Cholangiography

1. Differential diagnosis of obstructive and non-obstructive jaundice.
2. Diagnosis and location of a carcinoma of the biliary tract; determination of the site of the obstruction; preoperative selection of the type and location of the biliary shunt to be performed.
3. Determination of the exact number and location of calculi in the biliary tract.
4. Delineation of an anatomical abnormality in the biliary tract:
   (a) congenital: biliary tract atresia, choledochal cyst, etc.
   (b) acquired: stenosis, stricture, etc.
5. Investigation of some obscure cases of post-cholecystectomy syndrome.
6. Accurate determination of the origin of some persistent external biliary fistulas.
7. Decompression (by means of the polythene tube) of the biliary tract pending operation.
8. The procedure permits preoperative cytological, bacteriological and chemical study of the bile.

ods because the required concentration of contrast medium can be supplied on demand and under direct vision. If the information obtained by this examination has been obtained beforehand, a state of relaxation prevails in the operating-room in marked contrast to the tension which often accompanies the performance of auxiliary procedures during major operations.

In patients with hepatitis it frequently happens that no bile can be aspirated. Such a negative result contraindicates surgery. Laparotomy which is always undesirable in such patients can therefore be avoided. Flemma *et al.*,[1] of Durham, reported 47 patients in whom percutaneous transhepatic cholangiography was carried out. In six of these patients the biliary tract could not be visualized; none of the six required surgical exploration; the final diagnosis was hepatitis. Prioton, Vialla and Pous[8] noted that no bile was withdrawn in patients with hepatitis. Kaplan *et al.*[19] reported the results of percutaneous transhepatic cholangiography in 40 patients; 10 were negative; laparotomies were performed upon these 10 patients and revealed that eight had hepatitis and two had extrahepatic block. Royer *et al.*[14] reported the results of percutaneous transhepatic cholangiography in 19 patients, of which four were negative; at laparotomy these four patients had hepatitis. It therefore appears that when percutaneous transhepatic cholangiography is carefully attempted but no visualization is obtained, hepatitis is present.

Radiological Features

Certain radiological features that may be noted during cineradiography in percutaneous transhepatic cholangiography are worthy of comment.

If there is *neoplasia of the head of the pancreas*, an increase may be noted in the diameter of the common bile duct, which appears to be greatly dilated and tortuous. The distal extremity of the common bile duct is cup-shaped, with a distal convexity and some irregularity of the contours; the contrast medium does not penetrate into the duodenum (Fig. 9).

In cases of *choledocholithiasis with obstruction of the ampulla of Vater*, the common bile duct may be dilated, but usually less so than in pancreatic neoplasia. The boundary of the shadow produced by the contrast medium, which follows the contour of the stone, has a convexity in the direction of the proximal end of the common bile duct. The contrast medium passes into the duodenum, except when there is complete obstruction of the common bile duct by the stone (Fig. 5).

In mild *choledochal stricture*, percutaneous transhepatic cholangiography is the diagnostic method of choice. It permits the determination of the type of repair or plastic procedures which should be considered (Fig. 7).

Case Reports

Case 1.—Mrs. B. (Fig. 5), a 43-year-old woman, was admitted to Notre Dame Hospital, Montreal, because of generalized jaundice of eight days' duration. It developed a few days after the onset of pain in the right hypochondrium, which was referred to the angle of the right scapula. Laboratory tests indicated obstructive jaundice. Routine gallbladder films showed no opacification of the biliary tract, and percutaneous transhepatic cholangiography was performed on August 7, 1963. Injection into the bile duct gave good opacification of the intrahepatic biliary tract. The intrahepatic duct system, the common hepatic duct and the common duct were well visualized. The common duct was large and sinuous, and at the distal end a sharply defined shadow was seen suggestive of a stone of considerable

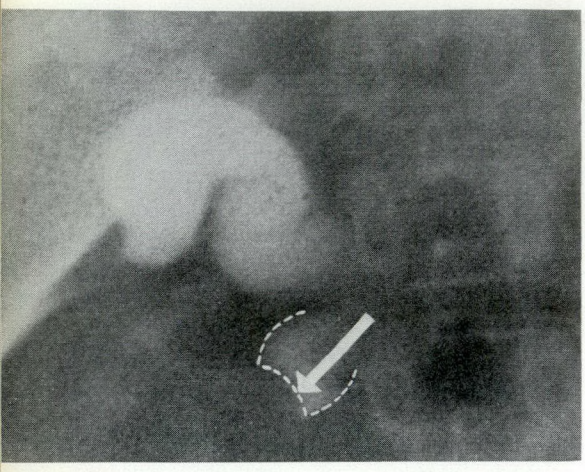


Fig. 5.—Stone in the common duct which is dilated. Note the convexity in the direction of the proximal end of the common duct (magnification of a 16 mm. film).

size. Cholecystectomy-choledochotomy, performed a few days later, revealed the presence of an enormous stone that completely blocked the distal common duct. The stone was removed and a T-tube put in place. The postoperative course was uneventful.

CASE 2.—Mrs. S. (Fig. 6), an 82-year-old woman, was admitted because of progressive jaundice of approximately two months' duration, accompanied by attacks of abdominal pain, nausea and vomiting. The urine was dark and the stools were pale. Laboratory tests pointed to obstructive jaundice; routine radiography showed no opacification of the biliary tract. Percutaneous transhepatic cholangiography performed on August 16, 1963, gave good opacification of the hepatic ducts and common bile duct, which were considerably dilated. A large intracholedochal stone was observed in the distal third of the duct. On increasing the pressure during injection of the medium, a small quantity of the latter passed on each side of the stone into the second portion of the duodenum. Cholecystectomy, choledochotomy and removal of the stone were carried out without difficulty.

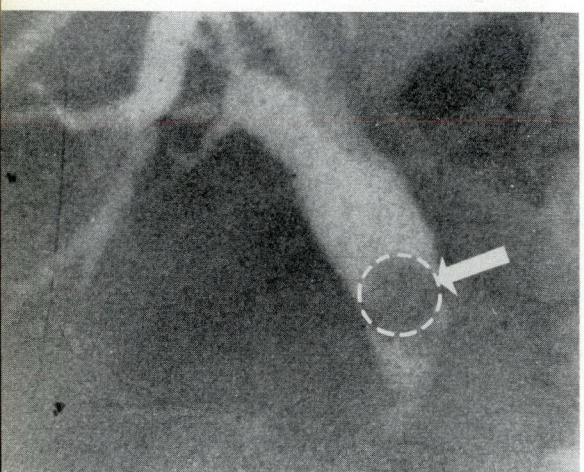


Fig. 6.—Stone in the common duct which is dilated. Obstruction is not complete and contrast medium flows past the stone (magnification of a 16 mm. film).

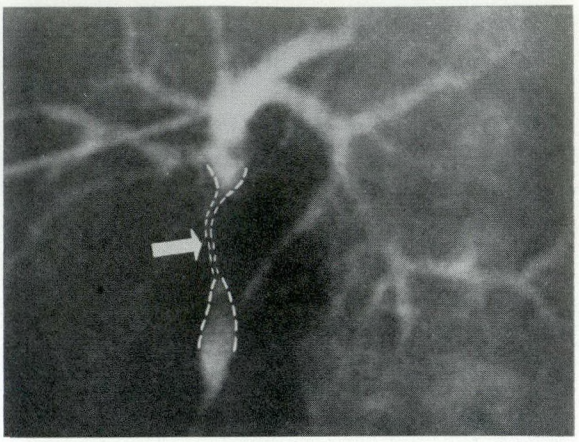


Fig. 7.—Post-cholecystectomy stenosis. In the upper left-hand corner one sees the needle in the hepatic hilum (magnification of a 16 mm. film).

CASE 3.—Mrs. D. (Fig. 7), a 64-year-old woman, had advanced biliary cirrhosis, and a history of cholecystectomy seven years before. In 1960, she had jaundice accompanied by fever and chills. Choledochotomy for choledochal stricture was performed in April 1961 and choledochoplasty in October 1961. In 1963 another episode of jaundice occurred with chills and fever, accompanied by melena and hematemesis owing to esophageal varices. The stools, however, were well pigmented. In July 1963, percutaneous transhepatic cholangiography was carried out. The bile ducts were not dilated, but there was a clearly defined stricture about 3 cm. long in the central portion of the choledochus. A splenorenal shunt was performed. The postoperative course was satisfactory and an hepaticojejunostomy is being considered for the near future.

CASE 4.—Mrs. B. (Fig. 8), a 57-year-old woman, was admitted because of pruritis, pale stools, dark urine and weight loss of 15 lb. in three months. Laboratory tests pointed to obstrucive jaundice and on routine radiography no opacification of the biliary tract was

seen. Percutaneous transhepatic cholangiography, performed on August 16, 1963, showed dilated bile ducts; the common duct opacified slowly and with difficulty, but was not dilated. However, it was *pushed back* toward the median line and neoplasia of the gallbladder was suspected. Cholecystectomy and choledochotomy were performed on the same day. At operation, a carcinoma of the gallbladder was found compressing the common duct. Transhepatic cholangiography helped the surgeon in dissecting out the common duct because he knew in advance where to find it. Only a cholecystectomy and a choledochotomy using a T-tube could be performed because there were metastases in both lobes of the liver. The patient's jaundice and pruritis cleared in a few days.

CASE 5.—Mr. H. (Fig. 9), a 58-year-old man, was admitted with progressive jaundice dating back one month, which was accompanied by severe pruritus. Pain was not a symptom and there was no tenderness on examination. The stools were pale and the urine was dark. The patient had lost 15 lb. in two months. Laboratory data pointed to obstructive jaundice. No opacification of the biliary tract was obtained on routine radiography. Percutaneous transhepatic cholangiography, performed on August 15, 1963, gave good visualization of the intrahepatic ducts and gallbladder. Both hepatic ducts and the common duct were well visualized. However, the common duct was considerably enlarged and its lumen was obliterated at the junction of the middle and distal thirds. The line of demarcation was irregular and jagged and suggested blocking by pancreatic neoplasia with invasion of the common bile duct. An operation was performed the same day and revealed neoplasia of the head of the pancreas. Cholecystojejunostomy and enteroenterostomy were carried out.

CASE 6.—Mr. B. (Fig. 10), a 35-year-old man, was admitted because of gastrointestinal complaints. He had lost 40 lb. in five months. Severe jaundice was present; the stools were clay-coloured and the urine was dark. Laboratory tests pointed to obstructive jaundice, and routine radiography gave no visualization of the bile ducts. Radiography after a barium meal revealed an "amputation" shadow at the level of the pyloric antrum and extrinsic compression of the internal aspect of the first, second and third portions of the duodenum. Percutaneous transhepatic cholangiography performed in April 1963 gave good visualization of the intrahepatic ducts, which were

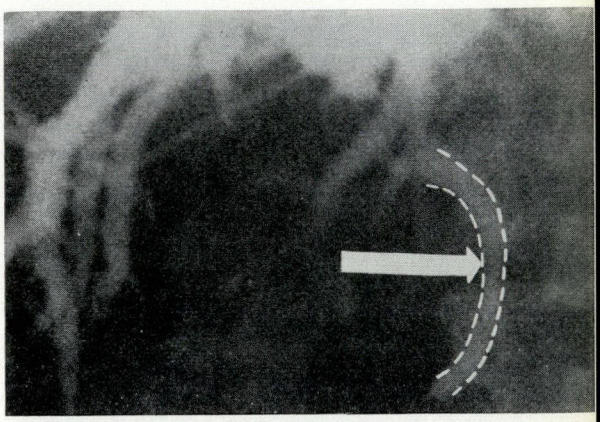


Fig. 8.—Normal common duct pushed aside by a gallbladder tumour (magnification of a 16 mm. film).

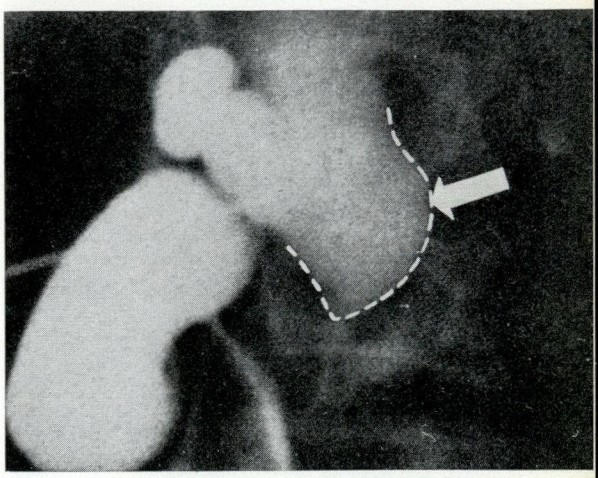


Fig. 9.—Neoplasia of the head of the pancreas. One sees the important dilatation of the common duct and the distended gallbladder (Courvoisier's sign) in the lower left-hand corner (magnification of a 16 mm. film).

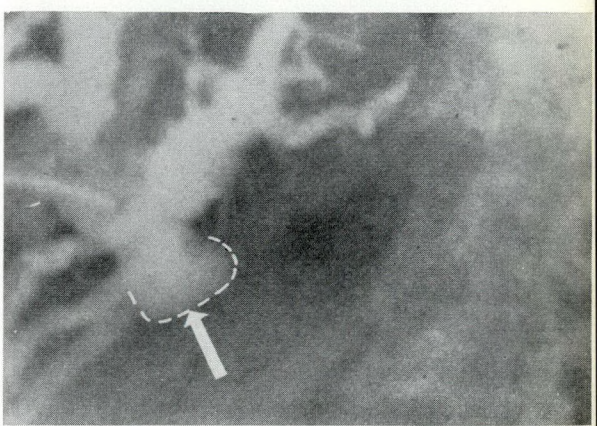


Fig. 10.—Invading neoplasia of the head of the pancreas. Note the distal convexity of the cupule and the needle in the hepatic hilum (magnification of a 16 mm. film).

greatly dilated up to the confluence of the two hepatic ducts at the beginning of the common bile duct. In dextroanterior oblique projection, the common-duct stump appeared to be dilated and flattened and to have an irregular contour. It was concluded that the common duct was blocked fairly high up by direct invasion by tumour, presumably of pancreatic origin. At operation there was an almost complete pyloric obstruction by an enormous pancreatic tumour, which compressed and invaded the common duct and pylorus. Cholecystojejunostomy and enteroenterostomy were performed.

CASE 7.—Mrs. D., a 54-year-old woman, was admitted to the Department of Surgery because of jaundice, which had started a fortnight earlier. Laboratory analyses pointed to obstructive jaundice, and routine radiography furnished no useful information. Percutaneous transhepatic cholangiography performed on May 14, 1963, gave good visualization of the intrahepatic ducts which were greatly dilated. The common hepatic duct was completely occluded at a point about 2 cm. from its origin at the junction of the two hepatic ducts. This "amputation" picture was complete and convex inside the hepatic duct, which suggested the presence of a tumour or of an enormous calculus at this point. An operation performed a few days later revealed neoplasia of the biliary tract with generalized metastases.

CASE 8.—Mr. M., a 75-year-old man, was admitted because of acute abdominal pain, nausea, vomiting, anorexia and attacks of syncope. The patient was febrile and his general condition was poor. Laboratory analyses did not suggest biliary tract obstruction. There was a leukocytosis of 12,700 per c.mm., neutrophilia of 88% and a sedimentation rate of 29 mm. in 1 hour. All liver function tests were normal with the exception of bromsulphalein (BSP) of 16% (upper limit of normal). The gallbladder was not visualized on cholecystography, and the extrahepatic bile ducts were not visualized by intravenous cholangiography. Percutaneous transhepatic cholangiography was performed on November 8, 1963. The trocar reached the gallbladder through the gallbladder bed and dirty cloudy bile was withdrawn. The biliary tract was easily injected from the gallbladder. Bile culture showed the presence of *E. coli* which was found to be sensitive to kanamycin and colimycin. Cholecystectomy for pyocholecystitis was performed the same day. No bile was found in the abdomen. The postoperative course was uneventful.

CASE 9.—Mr. P., a 56-year-old man, was admitted on November 19, 1963. Palliative gastrectomy had been performed in 1960 for epithelioma of the gastric antrum. He had lost 20 lb. during the previous two to three months and had gastrointestinal complaints. Jaundice had begun one week previously with clay-coloured stools and dark urine. Laboratory findings pointed to obstructive jaundice and an iron-deficiency anemia. Routine radiography did not visualize the hepatic ducts. Percutaneous transhepatic cholangiography gave good visualization of the hepatic ducts which were only slightly dilated. The middle third of the choledochus was extrinsically compressed, but nevertheless the contrast medium readily passed into the duodenum. On November 29, 1963, a recurrence of gastric neoplasia with invasion of the distal common duct was demonstrated at laparotomy. Cholecystojejunostomy with jejunojejunostomy was performed.

SUMMARY

The theoretical and practical values of percutaneous transhepatic cholangiography are discussed. The history of the procedure is reviewed and the technique adopted by the authors is described. The present indications for the use of this diagnostic method are given, particularly its use in the differential diagnosis of obstructive and non-obstructive jaundice in patients where other means of investigation have been unsuccessful in establishing the diagnosis.

The simplicity and safety of this technique is emphasized. Nevertheless it should be performed in a surgical centre and it should not be considered as a routine examination.

The numerous advantages of this diagnostic method are brought out and the various complications (choleperitoneum, hemoperitoneum, pneumothorax, Gram-negative septicemias) described in the literature can be avoided by closely following the technique described (including the polythene drainage tube) and combining it with competent nursing.

Percutaneous transhepatic cholangiography can be performed upon jaundiced patients.

Nine cases studied by this method in the authors' department are described.

REFERENCES

1. FLEMMA, R. J. *et al.*: Percutaneous transhepatic cholangiography in the differential diagnosis of jaundice, *Surg. Gynec. Obstet.*, **116:** 559, 1963.
2. PRIOTON, J. B.: La cholangiographie par ponction extra-péritonéale du foie, *Presse Méd.*, **68:** 2308, 1960.
3. HOUSSET, E. AND VANTSIS, G.: La cholangiographie trans-pariéto-hépatique; à propos de 9 observations, *Presse Méd.*, **65:** 772, 1957.
4. VARELA FUENTES, B., BERTONI, C. AND POLERO, J.: La cholangiografia por puncion hépatica, *Prensa Méd. Argent.*, **44:** 2873, 1957.
5. ARNER, O., HAGBERG, S. AND SELDINGER, S. I.: Percutaneous transhepatic cholangiography: puncture of dilated and nondilated bile ducts under roentgen television control, *Surgery*, **52:** 561, 1962.
6. CARTER, R. F. AND SAYPOL, G. M.: Transabdominal cholangiography, *J. A. M. A.*, **148:** 253, 1952.
7. HUARD, P. AND DO-XUAN-HOP: La ponction transhépatique des canaux biliaires, *Bull. Soc. Med. Chir. de l'Indochine*, **15:** 1090, 1937.
8. PRIOTON, J. B., VIALLA, M. AND POUS, J. G.: Nouvelle technique de cholangiographie trans-pariéto-hépatique, *J. Radiol. Electr.*, **41:** 205, 1960.
9. CRISMER, R.: Cholangiographie transcutaneo-hépatique, *Acta Gastroent. Belg.*, **16:** 762, 1953.
10. DE LA FUENTE, R. *et al.*: Exploration des voies biliaires au moyen de la cholangiographie transabdominale, *Lyon Chir.*, **49:** 957, 1954.
11. HEPP, J. AND VAYRE: La cholangiographie transhépatique, *Rev. Int. Hépat.*, **9:** 633, 1959.
12. LÉGER, L., ZARA, M. AND ARVAY: Cholangiographie et drainage biliaire par ponction trans-hépatique, *Presse Méd.*, **60:** 936, 1952.
13. LÉGER, L., ZARA, M. AND WARGNIER, M.: Cholangiographie et drainage biliaire par ponction trans-pariéto-hépatique, *Arch. Mal. Appar. Dig.*, **42:** 967, 1953.
14. ROYER, M. *et al.*: La cholangiographie trans-pariéto-hépatique sous contrôle radioscopique, *Presse Méd.*, **64:** 976, 1956.
15. SANTOS, M., FIGUEROA, L. AND LOPEZ, O.: Percutaneous transhepatic cholangiography in the diagnosis of posthepatic jaundice, *Surgery*, **48:** 295, 1960.
16. SHALDON, S., BARBER, K. M. AND YOUNG, W. B.: Percutaneous transhepatic cholangiography; a modified technique, *Gastroenterology*, **42:** 371, 1962.
17. ATKINSON, M., HAPPEY, M. G. AND SMIDDY, F. G.: Percutaneous transhepatic cholangiography, *Gut*, **1:** 357, 1960.
18. GLENN, F. *et al.*: Percutaneous transhepatic cholangiography, *Ann. Surg.*, **156:** 451, 1962.
19. KAPLAN, A. A. *et al.*: Percutaneous transhepatic cholangiography, *Ann. Intern. Med.*, **54:** 856, 1961.
20. ISLEY, J. K., JR. AND SCHAUBLE, J. F.: Interpretation of the percutaneous transhepatic cholangiogram, *Amer. J. Roentgen.*, **88:** 772, 1962.
21. ROYER, M., SOLARI, A. V. AND LANARI, R. L.: La colangiografia no quirurgica; nuevo método de exploracion de las vias biliares, *Arch. Argent. Enferm. Apar. Dig.*, **17:** 368, 1942.
22. MUELLER, W. AND BURCKHARDT, H.: Puncture of gallbladder, *Deutsch. Z. Chir.*, **162:** 168, 1921.

RÉSUMÉ

Les auteurs étudient l'intérêt théorique et pratique de la cholangiographie per-cutanée transhépatique. Ils passent en revue l'historique de cet examen et décrivent leur technique. Ils précisent les indications actuelles de cette méthode de diagnostic, notamment: la différenciation du caractère obstructif ou non de l'ictère, dans les cas où les autres moyens d'investigation ne peuvent préciser le diagnostic. Ils insistent sur la simplicité et l'innocuité de cette technique qui doit cependant être réservée à un centre chirurgical: il ne s'agit pas ici d'un examen de routine. Ils mettent en valeur les nombreux avantages de cette méthode de diagnostic et discutent ses complications (cholépéritoine, hémopéritoine, pneumothorax, septicémies à Gram négatif) évitables si l'on associe à une technique rigoureuse (le polythène pour drainage de sécurité) des soins compétents. Cet examen peut être pratiqué chez un patient non ictérique. Une série de cas traités par cette méthode dans le service des auteurs est résumée. Cette publication est une introduction à une étude clinique beaucoup plus vaste actuellement en cours à l'Hôpital Notre-Dame de l'Université de Montréal.

TIBIAL PLATEAU FRACTURES

T. W. BARRINGTON, M.D., F.R.C.S.[C]* and
F. P. DEWAR, M.D., F.R.C.S., F.R.C.S.[C],† *Toronto, Ont.*

As ONE of the facets of a continuing study of fractures of the tibia, a review of tibial plateau fractures has been carried out by the Workmen's Compensation Board of Ontario. During the years 1959 and 1960, 147 claims involving fractures of the tibial plateau were settled by the Workmen's Compensation Board. One hundred and forty-two of these occurred in men and five in women. The left side was affected in 80 and the right in 64. Fractures were bilateral in three. The average age at the time of injury was 48 years.

Sixty-five of these patients were personally interviewed, examined and radiographed in this series of 147, i.e. 44.2%. The remainder, 82, were not reviewed for reasons outlined in Table I.

TABLE I.—REASONS FOR NON-REVIEW —82 PATIENTS

		No. of patients
Moved, unable to locate		40
Disinterested, busy, unable to get off work		23
Geographically inaccessible areas		12
Deceased:		
from unrelated causes	6	
from bronchopneumonia related to crushed chest	1	7

Cotton and Berg[1] described fracture of the lateral tibial plateau as a "bumper" or "fender" fracture. This term hardly seems applicable in this group since only six patients in this series were struck by a moving vehicle. The greatest number of injuries were sustained in falls from heights (Table II).

In workmen, fracture of the tibial condyle was not a solitary injury in 18% of cases. Twenty-seven men had 41 other injuries (Table III), suggesting that the degree of trauma producing injury was considerable.

TABLE II.—MECHANISM OF INJURY —147 PATIENTS

	No. of patients
Fall from ladder, heights, etc.	43
Struck glancing blow by falling or rolling object	38
Slips, falls	23
Direct blow	14
Crushing injury	12
Passenger in a moving vehicle	11
Struck by moving vehicle	6

Seventy-four months was the average time of follow-up in this series and ranged from 44 to 156 months.

TABLE III.—ASSOCIATED INJURIES: PRINCIPALLY FRACTURES

Tibia	10	Shoulder	1
Ribs	6	Olecranon	1
Wrist	5	Dislocation hip	1
Spine	3	Mandible	1
Pelvis	3	Facial nerve palsy	1
Os calcis—bilateral	2	Burns	1
Ankle	2	Inguinal hernia	1
Metatarsals	2	Concussion	1

It is generally accepted that valgus strains lead to fractures of the lateral condyle and that varus strains, which are much less common, will produce a fracture of the medial tibial plateau. This same mechanism plus an excessive axial load in the line of the tibia will result in a T-shaped or Y-shaped fracture of the proximal end of the tibia.

The classification of injuries used in this series is modified from Palmer.[2]

Type I: Split Fractures Lateral Condyle (Fig. 1)

The lateral edge of the lateral femoral condyle is driven down into the articular surface of the lateral tibial condyle, producing a wedge-shaped fragment of varying size which becomes displaced outwards. There is seldom any downward displacement.

*Workmen's Compensation Board Fellow, Division of Orthopedic Surgery, Toronto General Hospital (1963-1964); Attending Staff, Orthopedic Division, Toronto East General and Orthopaedic Hospital, Toronto.
†Division of Orthopedic Surgery, Toronto General Hospital.

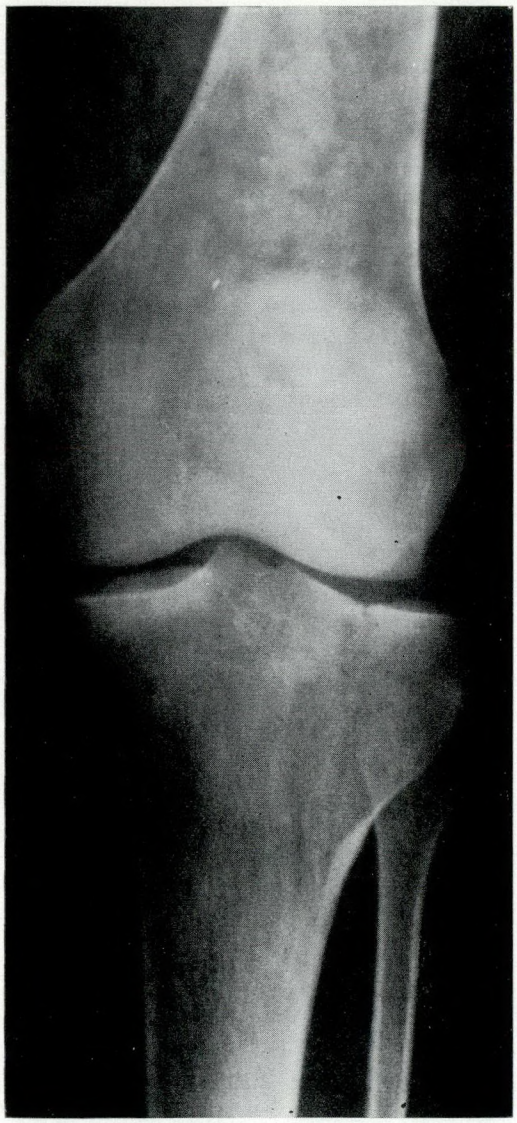


Fig. 1.—Type I injury. Split fracture of lateral condyle. A wedge-shaped fragment is displaced outwards. There is seldom any downward displacement.

Type II: Split Fractures of Medial Condyle (Fig. 2)

Similar to Type I, but on the medial side.

Type III: Compression Fractures (Figs. 3a and b)

This type is produced when the blunt inferior articular surface of the femoral condyle is driven into the articular surface of the apposing tibial condyle, acting like a hammer in contrast to the chisel-like action in Types I and II. The displacement ranges from depression of the anterior, posterior, lateral or medial part of the plateau with comminution of the area and depression of the whole condyle.

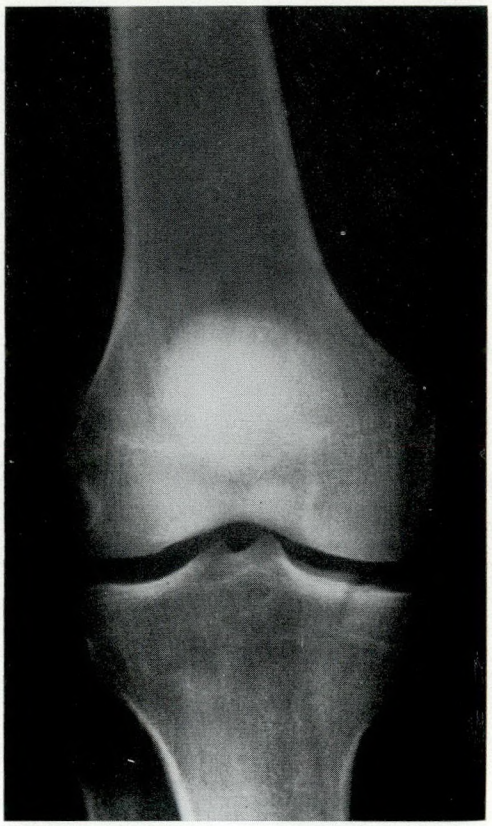
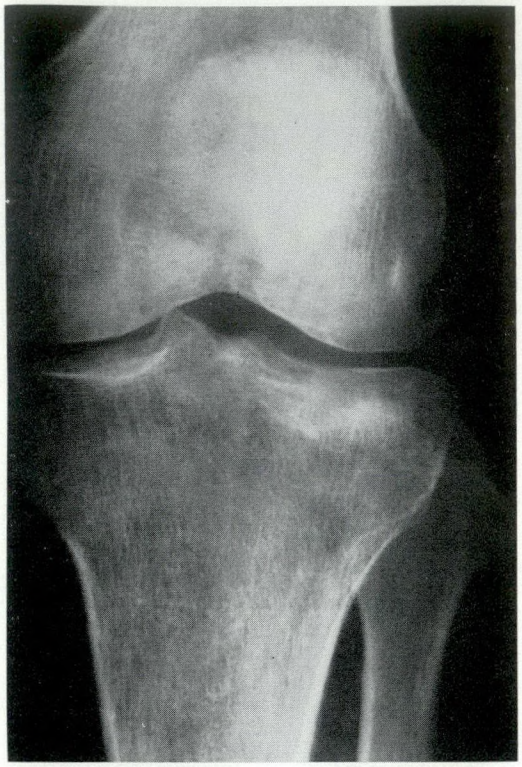


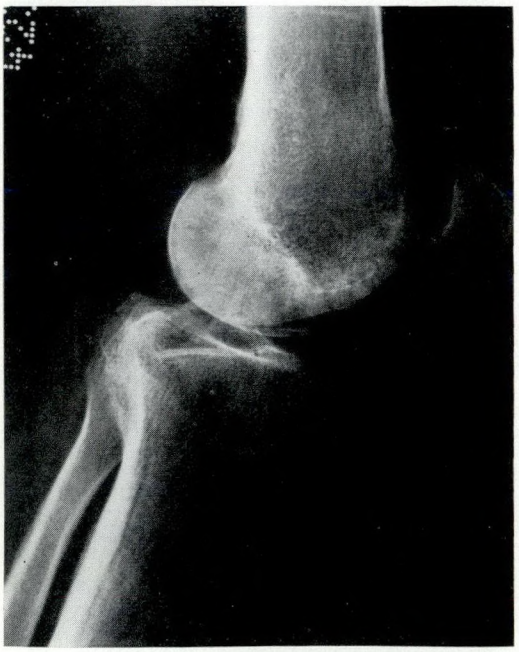
Fig. 2.—Type II injury. Split fracture of medial condyle. A wedge-shaped fragment is displaced outwards.

Type IV: Compression Fracture (Fig. 4)

A large lateral fragment is split off and displaced outwards, whereas the central and medial part of the articular surface is comminuted and displaced obliquely downwards and inwards. Frequently fragments from the central part of the articular surface are driven downwards and wedged in the split between the major fragments. This type is often associated with fracture of the neck of the fibula.



(a)



(b)

Fig. 3.—Type III injury (anteroposterior and lateral). Depression of (a) central and (b) posterior aspect of lateral tibial plateau produced by hammer-like action of apposing tibial condyle.

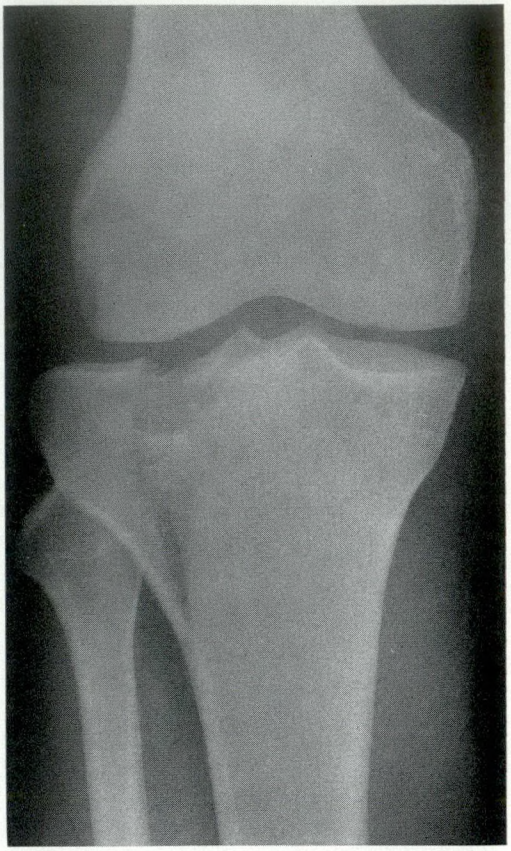


Fig. 4.—Type IV injury. Lateral fragment is split off and displaced outwards and central fragment is displaced downwards.

Type V: T-Shaped or Y-Shaped Fracture

Both the lateral and medial condyles are fractured and the predominant force is vertical.

(a) *Fig. 5.*—Both condyles are fractured and displaced downwards and outwards—each as one large fragment, and the upper tibial shaft is driven between the articular surfaces. Often the articular surfaces in this type of fracture show very little damage.

(b) *Fig. 6.*—Both condyles are fractured and displaced outwards and downwards, but one condyle shows features of Types III and IV, i.e. comminution.

The incidence of each type of fracture is outlined in Table IV. The commonest types of injury are Types III and I. Injuries of the medial condyle occurred in 12% of all injuries.

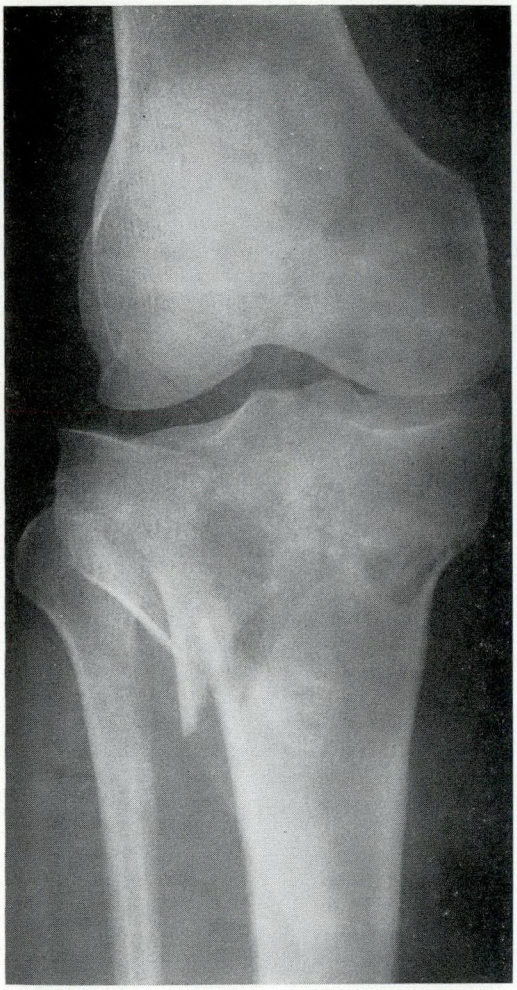


Fig. 5.—Type V(a) injury. Both condyles fractured without much comminution.

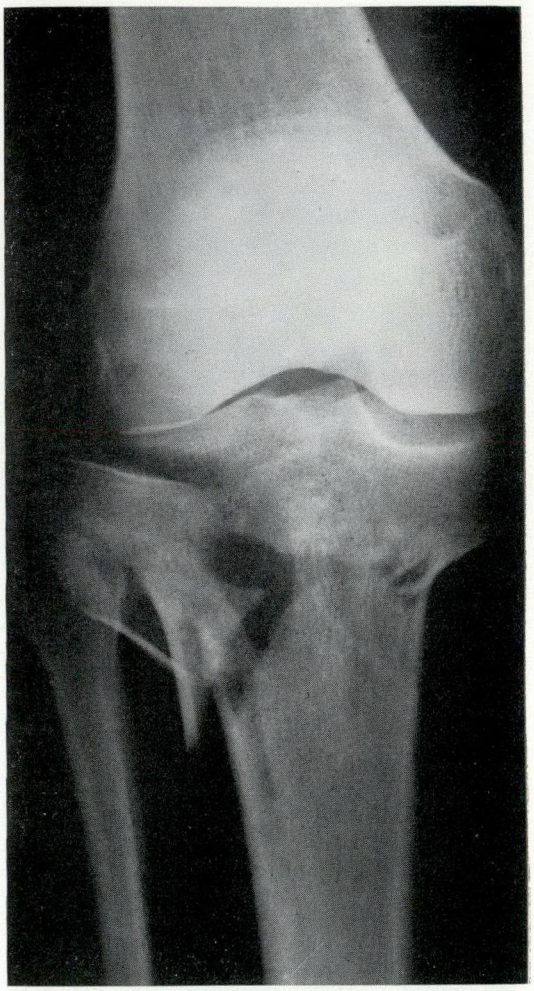


Fig. 6.—Type V(b) injury. Both condyles fractured and displaced outwards. Marked comminution of lateral condyle.

Initially, these injuries were cared for principally by general practitioners, but in all instances when the injury was considered to be of a serious nature, the responsibility and care was transferred to a general or orthopedic surgeon.

Forty-one of the 147 patients were operated upon early as a primary form of treatment (28%). The time lapse between injury and operation varied from six hours to 28 days. The average delay in operation was seven days. The reasons for delay in performing the operation, when recorded, are given in Table V.

TABLE IV.—Incidence of Fracture Types—147 Patients

Type	*No. of patients*	*%*
I	39	27
II	18	12
III	46	31
IV	14	9
V (a)	20	14
(b)	10	7

TABLE V.—Reasons for Delay in Operation

	No. of patients
Severe associated injuries	5
Unrecognized injuries	5
Shock	1

Of the 65 patients reviewed at follow-up, 24 were operated upon early. The operative procedures carried out are as listed in Table VI.

TABLE VI.—Early Surgical Procedures

Arthrotomy, meniscectomy with or without elevation and bone graft	10
Screw or wire fixation with or without elevation and bone graft	7
Manipulation and fixation with a Barr bolt	5
Tibial tubercle traction	4

In some instances, two procedures were carried out on the same knee.

Late surgical procedures included arthrodesis, two; removal of a loose bone fragment, one; insertion of Teflon prosthesis, one; and sympathectomy, one.

Complications during the first hospital admission were common, occurring in 18% of the 147 patients.

TABLE VII.—Complications During Early Active Treatment

Pulmonary embolism	5
Thrombophlebitis	4
Volkman's ischemic contracture	4
Wound infection	3
Peroneal nerve lesion	2
Fat embolism	1
Thrombosis—external iliac artery	1
Nephrolithiasis	1
Reflex sympathetic dystrophy	1
Fracture of os calcis owing to fall at four months	1
Epididymitis	1
Bronchopneumonia (death)	1

The commonest complications were pulmonary embolism, five, and thrombophlebitis, four. Volkman's ischemic contracture occurred to a greater or lesser degree in four cases and wound infection in three cases. A total of 25 separate complications occurred in 23 patients (Table VII).

Results

In the assessment of results the criteria used conform to those of Palmer.[2]

Excellent.—Pain-free, full function, normal or almost normal mobility, stable, no atrophy.

Good. — Pain-free or occasional aching after activity, good mobility, moderate atrophy, increased crepitation as compared with the other side, stable.

Fair. — Inconvenience, limiting the working capacity, reduced mobility, atrophy, deformity, marked crepitation, slight instability.

Poor. — Rest pain, poor mobility or ankylosis, deformity, or gross instability.

TABLE VIII.—Late Clinical Results of Those Reviewed—65 Patients

Type	*No. of patients*	*Excellent*	*Good*	*Fair*	*Poor*
I	16	4	4	6	2
		50%		50%	
II	4	1	2	1	0
		75%		25%	
III	24	8	9	6	1
		70%		30%	
IV	6	0	3	3	0
		50%		50%	
V (a)	10	0	1	7	2
		10%		90%	
(b)	5	0	0	3	2
				100%	
Total	65	13	19	26	7

A careful attempt was made to correlate radiological findings at the follow-up examination with the clinical result, but no consistent relationship was seen. Commonly, an excellent clinical result was radiologically fair or poor, and the converse also held true.

TABLE IX.—Late Results of Conservative Treatment—41 Patients

Type	*No. of patients*	*Excellent*	*Good*	*Fair*	*Poor*
I	8	4	1	2	1
		63%		37%	
II	2	0	1	1	0
		50%		50%	
III	20	7	9	3	1
		80%		20%	
IV	2	0	1	1	0
		50%		50%	
V (a)	7	0	0	5	2
				100%	
(b)	2	0	0	1	1
				100%	
Total	41	11	12	13	5

The results are assessed in Tables VIII, IX and X. The overall results were: excellent, 18%; good, 31%; fair, 40%; and poor, 11%. The least satisfactory results overall were in Types V(a) and (b) and generally the greater the magnitude of injury the less satisfactory was the end-result.

Satisfactory results were considered as being those which were classed as excellent or good. Unsatisfactory results were fair or poor.

Although the number of cases reviewed is small it can be seen that Type I and Type III fractures, managed by conserva-

TABLE X.—Late Results of Operative Treatment—24 Patients

Type	No. of patients	Excellent	Good	Fair	Poor
I	8	0	3	4	1
		38%		62%	
II	2	0	2	0	0
		100%			
III	4	1	1	2	0
		50%		50%	
IV	4	0	1	3	0
		25%		75%	
V (a)	3	1	0	1	1
		33⅓%		66⅔%	
(b)	3	0	0	2	1
				100%	
Total	24	2	7	12	3

tive measures, resulted in satisfactory results in 63% and 80% respectively, whereas the same types which were operated upon had satisfactory results in 38% and 50%. As the injury increased in severity resulting in Type IV and Type V injuries, operative measures did very little to improve the end-result. In those in whom meniscectomy was performed, there was a higher proportion (91%) of unsatisfactory results.

TABLE XI.—Late Complications—65 Patients

Barometric pain	28
Extensor lag more than 5°	14
Constant severe pain	13
Marked crepitus, chondromalacia patella	11
Valgus more than 5°	9
Flexion less than 90°	7
Instability	11
Wasting more than ½ inch	12
Anterior "drawer" sign Type III	3
Type IV	2
Type V (a)	3
Arthrodesis	2
Thrombophlebitis of long standing	3

At follow-up examination many complications were noted, several often being present in the same individual (Table XI).

Barometric pain was a common sequela. A torn or attenuated anterior cruciate ligament was present in eight patients, all of whom had more severe types of injuries. The medial collateral ligament was ruptured in two patients, both of whom had severe Type IV injuries. Both had a fair result and surgical repair was not carried out. Arthrodesis was carried out late upon two patients, one with Type V(a) and one with Type V(b) fractures, and the end-results were satisfactory.

TABLE XII.—Time of Immobilization Related to End-Result—65 Patients

Immobilization less than 6 weeks				*Immobilization more than 6 weeks*			
Excellent	*Good*	*Fair*	*Poor*	*Excellent*	*Good*	*Fair*	*Poor*
12	6	6	1	1	14	19	6
72%		28%		36%		64%	
25 patients				40 patients			

Table XII relates length of immobilization to end-results.

It would seem that early movement without weight-bearing produces a higher proportion of satisfactory results regardless of the type of injury (72% as compared with 36%, Table XII).

It was also noted that chondromalacia of the patella was a common complication in those immobilized over three months. Quadriceps wasting was also present in those immobilized for more prolonged periods. Thrombophlebitis was persistent in three patients, all of whom were immobilized for at least three months.

All patients began weight-bearing at approximately three months, whether an operation was performed or not.

The average time in hospital was 41 days, varying from zero to 43 weeks.

Eighty-one (55%) of the 147 patients were treated in the rehabilitation centre. Their average stay was 14 weeks and varied from two to 70 weeks.

TABLE XIII.

Type of injury	*No. of patients*	*Average time off work (months)*	*No. of patients*	*Pension award* (average) %*	*Finalized time† (months)*
I	39	6	12	7	15
II	18	9	6	10	13
III	46	7	15	9	15
IV	14	12	11	8	18
V (a)	20	10	16	10	26
(b)	10	15	8	26	31

*The percentage of the worker's salary awarded as a pension.
†Time required to settle the claim: treatment completed or pension awarded.

Table XIII relates the time off work, percentage award as pension and the time following injury that the case was settled.

From the point of view of time off work, Types IV and V(a) and (b) are the most severe types of injuries. This impression is borne out by the fact that 80% of these injuries resulted in awards varying from 8% to 26% of their salary. Furthermore, the claims in these injuries were settled after a longer period than were those in other types of injuries.

TABLE XIV.—Post-Injury Work History —147 Patients

	No. of patients	*%*
Lighter work	29	19
Unable to work	5	4
Retired	6	4
Deceased	7	5
Same work	100	68

Because of their injury, 19% of the patients were forced to return to a lighter type of employment (Table XIV). Four per cent were unable to return to work. Another 4% had retired at follow-up. Five per cent had died and 68% had returned to their usual full-time employment.

Discussion

Fractures of the tibial plateau are serious injuries resulting in prolonged hospital stay, prolonged use of rehabilitation centres and prolonged absence from work, often leading to a change in occupation. Associated injuries and complications during treatment are to be anticipated. Early mobilization will result in a diminution in the severity and number of complications.

In general, surgical measures have produced less satisfactory results than conservative measures. Types I and III injuries do better with manipulation and early mobilization. After reviewing these cases we feel that a Barr bolt, lag screw or wiring should be used to obtain stability only when there is a large depressed fragment without significant comminution. It is doubtful that arthrotomy and elevation of depressed fragments add anything to the clinical end-result in the more severely comminuted fractures, Types III and IV. Meniscectomy should not be performed as a primary procedure, as in this group of patients it resulted in a high proportion of unsatisfactory results. In Type V fractures one can expect uniformly unsatisfactory results, no matter which method of treatment is chosen. We feel that in this group of patients, as in those who must be returned quickly to heavy work, early arthrodesis should be considered rather than waiting to use it as a salvage procedure.

Summary

One hundred and forty-seven patients with tibial plateau fractures finalized by the Workmen's Compensation Board of Ontario during 1959 and 1960 have been reviewed. Sixty-five members of this group were personally reviewed. Associated injuries and early and late complications have been tabulated. The results of operative versus conservative measures are tabulated and discussed. A plea for early mobilization and, in general, more conservative measures of management is made in the hope of obtaining a higher proportion of satisfactory end results.

We wish to thank the Workmen's Compensation Board of Ontario for their generous assistance in this study.

References

1. Cotton, F. J. and Berg, R.: "Fender fractures" of the tibia at the knee, *New Eng. J. Med.*, **201:** 989, 1929.
2. Palmer, I.: Fractures of the upper end of the tibia, *J. Bone Joint Surg.* [*Brit.*], **33B:** 160, 1951.

Résumé

Ceci est une étude statistique, conduite par le "Workmen's Compensation Board of Ontario" des cas de fractures du plateau tibial qui ont été rapportées à cet organisme dans les années 1959 et 1960. On trouve ainsi une série de 147 cas, dont 142 chez des hommes; les fractures étaient à gauche dans 80 cas, à droite dans 64 et bilatérales dans trois. L'âge moyen des malades était 48 ans. Soixante-cinq de ces patients purent être réexaminés, radiographiés et interrogés. Les autres ne le furent pas pour des raisons diverses: impossibilité de retrouver l'adresse, incapables de venir à la convocation, morts de causes diverses (sans relation avec la fracture). Les causes des fractures étaient, par ordre de fréquence: chute d'une échelle, traumatisme par objet en mouvement, glissades, contusion directe, circulation automobile (passager dans une voiture ou piéton). Les lésions furent cataloguées selon Palmer: (1) arrachement net du condyle externe: 27%; (2) arrachement net du condyle interne: 12%; (3) fracture par compression: ici les condyles fémoraux agissent comme un marteau et tendent, sous l'action d'un traumatisme, à défoncer le plateau tibial; ce groupe comprend 31% des cas; (4) un second type de fracture par compression où il existe un large fragment externe déplacé en dehors, cependant que les régions centrale et interne du plateau sont déplacées en dedans et en bas; représentant 9% des cas; (5) fractures en T ou en Y: 21%. Les complications furent très fréquentes dans les premiers jours d'hospitalisation (18%), comprenant les embolies pulmonaires, les thrombophlébites, la contracture de Volkman, les infections etc. Les traitements furent le plus souvent conservateurs et relativement peu d'interventions furent faites immédiatement. Les resultats éloignés sont étudiés: dans l'ensemble, ces statistiques montrent que la guérison est meilleure avec le traitement conservateur qu'avec la chirurgie; ces chiffres sont discutés en détail.

GASTRIC COOLING AND HEPATIC FUNCTION*

N. B. HERSHFIELD, L.R.C.P.S.(I),† J. F. LIND, M.D., F.R.C.S.[C]‡ and J. A. HILDES, M.D., F.R.C.P.[C],§ *Winnipeg, Man.*

GASTRIC cooling has been recently introduced as a method of controlling upper gastrointestinal hemorrhage.[1, 2] The effects of such cooling on the function of adjacent organs, such as the liver, have not been reported in humans, but in our own small experience in using gastric cooling to control hemorrhage in patients with liver disease, liver function appears to deteriorate. Since it is well known that severe hemorrhage itself has an adverse effect on liver function,[3] it is clinically difficult to separate the contribution of this effect (hemorrhage) from other factors, including the possible adverse effects of cooling. The present study was undertaken to find out if gastric cooling had an adverse effect on liver function. Since cooling is achieved with a distended balloon in the stomach, the effect of the balloon itself must also be taken into account.

In studies on animals it has been reported that hepatic blood flow was depressed in some cases by the mere presence of a distended gastric balloon, and further depressed by gastric cooling.[4] Studies of the function of the pancreas and kidneys during gastric cooling have also been reported in animals.[5] Although in this study we were concerned mainly with hepatic function, some parameters of renal and pancreatic function have also been examined.

METHODS

Excretory function of the liver was determined using bromsulphalein (BSP). Five milligrams of the dye per kilogram body weight was injected intravenously three times at hourly intervals in each subject, and blood samples were taken from another vein before and at 15, 25 and 45 minutes after each injection. The BSP concentration was determined photometrically.

The test was carried out on 18 subjects, 20 to 50 years of age, who had no evidence of liver disease. Nine subjects acted as a control group and in these a gastric cooling balloon was not used. In the remaining nine a gastric balloon was placed in the stomach and distended to 500 ml. with 95% alcohol, which circulated through a "K thermia" unit.* The circulating fluid was warm (35° C. to 37° C.) for the first and third hours, and cooled to 5° C. during the second BSP test. Iodopyracet (Diodrast) was injected into the balloon to verify its position in the stomach. One patient was excluded because he developed nausea and vomiting early during gastric cooling. This may have been due to the procedure[1, 2] or to a reaction to BSP.[6, 7]

Venous blood samples were also taken for determination of serum amylase, blood urea nitrogen and hematocrit before the first warm period; at the end of the cooling period; and at the end of the second warm period.

RESULTS AND DISCUSSION

Repeated Injection of BSP

Table I shows that the values in Test 1 fell within the usually accepted range of normal for the standard clinical BSP test.

The concentration of dye remaining in the serum increased with each succeeding dose of BSP. The increases between Test 1 and Test 2 and between Test 2 and Test 3 were similar in magnitude, and were statistically significant (t test for paired data).

The increasing concentration implies a decreasing ability of liver to excrete the dye. However, as Mendeloff *et al.*[8] pointed out, the calculation of the clearance of the

*From the Clinical Investigation Unit, Winnipeg General Hospital, and the Departments of Surgery and Medicine, University of Manitoba, Winnipeg, Man.

Supported by a grant from the Medical Research Council of Canada.

†Research Fellow, Clinical Investigation Unit, Winnipeg General Hospital, 1963-1964.

‡Markle Scholar in Academic Medicine.

§Director, Clinical Investigation Unit, Winnipeg General Hospital, (to whom requests for reprints should be addressed).

*Gorman-Rupp Industries, Belleville, Ohio, U.S.A.

TABLE I.—Serial BSP Retention in Control Subjects

(BSP concentration in mg. per 100 ml. at 45 min. after each injection)

Subject	Test 1	Test 2	Test 3	Differences between tests 2 - 1	Differences between tests 3 - 2
1	0.89	2.35	2.94	+1.46	+0.59
2	0.38	1.18	1.76	+0.80	+0.58
3	0.33	0.68	1.24	+0.35	+0.56
4	0.13	0.14	0.42	+0.01	+0.28
5	0.56	0.59	0.80	+0.03	+0.21
6	0.29	0.59	0.99	+0.30	+0.40
7	0.14	0.18	0.22	+0.04	+0.04
8	0.44	0.78	1.16	+0.34	+0.38
9	0.13	0.18	0.22	+0.05	+0.04
Mean	0.36	0.74	1.08		
±S.E.	±0.08	±0.23	±0.02		
Mean differences ± S.E.				0.36 ±0.16 p<0.05	0.34 ±0.07— p<0.001

dye from the rate of disappearance is a more sensitive index of the ability of the liver to handle the dye. By this method the clearance of dye may be actually greater after a second dose. Therefore the serial samples taken after each injection were used to calculate the clearance of dye in our subjects.

Fig. 1 shows the average curves for the nine control subjects. The log concentration is plotted against time. If clearance were proportional to concentration, the decline should be linear, but this is not so. For our purposes the 30-minute clearance was calculated from the mean fall in concentration between 15 and 45 minutes.

These clearance rates are shown for the control subjects in Table II. The difference in disappearance rates between Test 1 and Test 2 indicates a faster clearance by the liver after the second dye injection. However, the comparison between the second

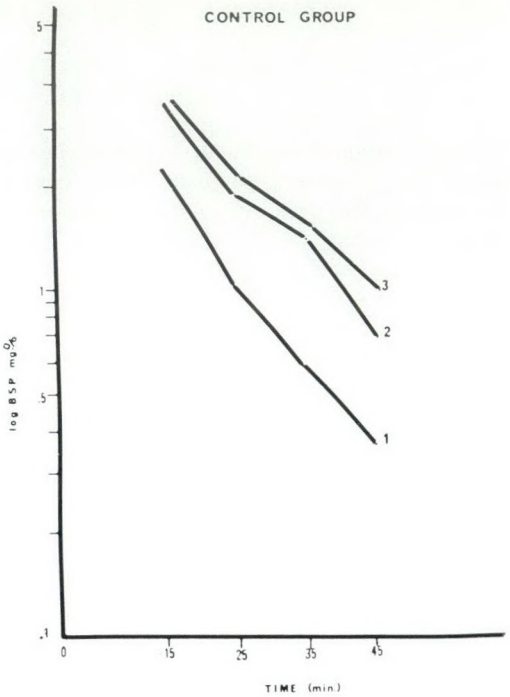


Fig. 1.—Control group: log mean BSP concentration plotted against time for Tests 1, 2 and 3.

and third injections indicates a slower clearance in the third test compared with the second. This may be interpreted as due to "saturation" of the hepatic mechanism for handling the dye, since the concentrations are higher in the third test. The latter finding is similar to the results after a second injection found in subjects with impaired liver function.[8]

In the test subjects the 45-minute concentrations after the first injection (Table III) are all well within the usually accepted range of normal for this test, and indeed are, on the average, lower than the comparable values in the control group, al-

TABLE II.—Disappearance Rate (D.R.) of BSP (mg. per 100 ml. per 30 min.) in Control Subjects

Subject	Test 1 15 min.	Test 1 45 min.	Test 1 D.R.	Test 2 15 min.	Test 2 45 min.	Test 2 D.R.	Test 3 15 min.	Test 3 45 min.	Test 3 D.R.	Differences in D.R. 2 - 1	Differences in D.R. 3 - 2
1.	2.71	0.89	1.82	4.92	3.50	2.57	5.74	2.94	2.80	+0.75	+0.23
2.	2.08	0.38	1.70	3.82	1.18	2.64	4.19	1.76	2.43	+0.94	—0.21
3.	2.01	0.13	1.88	3.10	0.18	2.82	2.34	0.22	2.12	+0.94	—0.74
4.	2.85	0.56	2.29	4.13	0.59	3.54	3.24	0.80	2.44	+1.25	—1.10
5.		0.33		3.40	0.68	2.72	4.00	1.24	2.76		—0.04
6.	1.07	0.13	0.94	1.86	0.14	1.72	1.92	0.42	1.50	+0.78	—0.22
7.	2.59	0.29	2.66	3.50	0.59	2.91	3.82	0.99	2.83	+0.25	—0.08
8.	2.01	0.14	1.87	3.10	0.18	2.82	2.34	0.22	2.43	+0.93	—0.21
9.	2.68	0.44	2.24	3.06	0.78	2.28	3.15	1.16	0.99	+0.04	—0.29
Mean D.R. ± S.E.			1.92 ± 0.2			2.66 ± 0.35			2.22 ± 0.29		
Mean differences in D.R. ± S.E.										0.74 ±0.12 p < 0.001	—0.29 ±0.13 p < 0.05

TABLE III.—Serial BSP Retention (mg. per 100 ml. at 45 min. After Each Injection) in Test Subjects

Subject	Test 1 warm	Test 2 cold	Test 3 warm	Differences between tests: 2 - 1	Differences between tests: 3 - 2
1	0.55	1.02	1.20	+0.42	+0.22
2	0.06	0.11	0.10	+0.05	−0.01
3	0.09	0.19	0.49	+0.10	+0.30
4	0.10	0.22	0.22	+0.12	0.00
5	0.25	0.38	0.60	+0.13	+0.22
6	0.37	0.92	0.89	+0.55	−0.03
7	0.50	0.80		+0.30	
8	0.09	0.16		+0.07	
Mean	0.25	0.48	0.56		
±S.E.	±0.07	±0.14	±0.16		
Mean difference ± S.E.				0.23 ±0.07	0.08 ±0.06
				p<0.01	p = 0.9

though not significantly so. These results are interpreted as indicating that the distended warm gastric balloon had no adverse effect on hepatic function. Similarly, the calculated clearances of BSP after the first dye injection in this group of subjects are, if anything, faster than the comparable clearances in the control group (Table IV).

The Effect of Gastric Cooling

Tables III and IV and Fig. 2 show the results of gastric cooling on a second injection of dye and the subsequent effect of gastric rewarming on the third injection.

As in the control subjects, the 45-minute dye concentrations in serum increased significantly between Test 1 and Test 2 (Table III). However, the 45-minute concentration in the third test, after the stomach had been rewarmed, showed only a small and insignificant increase compared with Test 2, in contrast to the controls. This suggests that rewarming had a beneficial

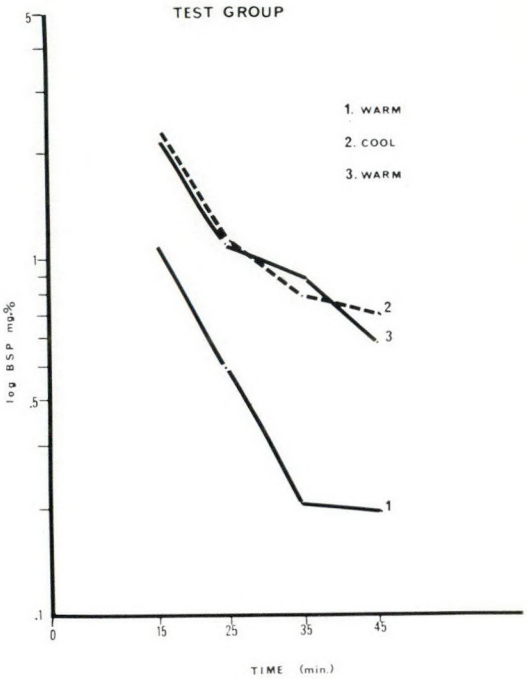


Fig. 2.—Test group showing the effect of gastric cooling in Test 2 by means of log BSP concentration plotted against time.

effect on hepatic function, and, by inference, that gastric cooling had an effect which is not apparent when only the 45-minute concentrations are examined.

The clearance of dye, which should be a more sensitive index of hepatic function under conditions of gastric cooling and rewarming, is shown in the appropriate columns of Table IV and also in Fig. 2. In spite of gastric cooling, the clearance rate in Test 2 was greater than in Test 1. This result is similar to that obtained in the control subjects and to Mendeloff's results in normal subjects and does not, in itself, lend any support to the hypothesis that gastric

TABLE IV.—Disappearance Rate (D.R.) of BSP (mg. per 100 ml. per 30 min.) in Subjects with Gastric Cooling

Subject	Test 1 "warm" 15 min.	Test 1 "warm" 45 min.	Test 1 "warm" D.R.	Test 2 "cool" 15 min.	Test 2 "cool" 45 min.	Test 2 "cool" D.R.	Test 3 "warm" 15 min.	Test 3 "warm" 45 min.	Test 3 "warm" D.R.	Cool-warm 2 - 1	Warm-cool 3 - 2
1.	1.18	0.55	0.63	2.18	1.02	1.16				+0.53	
2.	0.91	0.06	0.85	1.36	0.11	1.25	1.25	0.10	1.15	+0.40	−0.10
3.	1.03	0.09	0.94	1.69	0.19	1.50	2.86	0.49	2.37	+0.56	+0.87
4.	1.29	0.25	1.04	2.36	0.38	1.98	2.17	0.60	1.57	+0.94	−0.41
5.	2.68	0.10	2.58	3.07	0.22	2.85	2.15	0.22	1.93	+0.27	−0.92
6.	1.63	0.37	1.26	1.95	0.92	1.03	2.68	0.89	1.79	−0.23	+0.76
7.	1.03	0.09	0.94	1.87	0.16	1.71				+0.77	
8.	2.02	0.50	1.58	3.68	0.80	2.88				1.30	
Mean ± S.E.		1.19 ± 0.14			1.72 ± 0.25			1.76 ± 0.02			
Mean differences ± S.E.										+0.56 ±0.16	+0.04 ±0.01
										p < 0.01	p = 0.9

TABLE V.—The Effect of Gastric Cooling on Blood Urea Nitrogen Concentration, Hematocrit and Serum Amylase Concentration

Subject	*Parameter*	*Control*	*Post-cooling*	*Post-warming*
1	B.U.N. (mg. per 100 ml.)	17	16	15
	Hct. (%)	45	40	42
	Amylase (units per 100 ml.)	194	130	138
2	B.U.N.	19	18	17
	Hct.	45	44	45
	Amylase	148	160	133
3	B.U.N.	15	13	13
	Hct.	46	43	42
	Amylase	144	124	105
4	B.U.N.	14	12	10
	Hct.	44	41	46
	Amylase	184	184	157
5	B.U.N.	16	16	17
	Hct.	45	49	46
	Amylase	212	166	162

cooling has any effect on hepatic function in normal subjects. However, when the stomach is rewarmed during the third BSP injection, the "saturation" effect shown in the control subjects is not apparent since there is no significant difference between the second and third clearance tests. This again suggests that gastric rewarming had a beneficial effect on BSP clearance and, by inference, that gastric cooling may have had a small but undetected adverse effect.

It is possible that in patients with liver disease who undergo long periods of treatment, gastric cooling might depress hepatic function to a greater degree than is indicated in the above-noted normal subjects.

Effect of Gastric Cooling on Serum Amylase, Blood Urea Nitrogen, and Hematocrit

The results shown in Table V do not indicate any gross change in pancreatic and renal function in these subjects, although these are not sensitive tests of organ function.

Summary

Three serial BSP injections at hourly intervals in normal subjects showed an increased clearance of BSP following the second injection of dye, indicating an enhanced clearance at high blood concentrations. This is followed by reduced clearance, or "saturation", after the third injection in spite of a higher concentration.

A gastric balloon distended with 500 ml. of warm fluid (37° C.) had no effect on hepatic clearance of BSP in normal subjects.

A gastric balloon distended with 500 ml. of cold fluid (5° C.) may have had a slight effect on hepatic clearance since no "saturation" phenomenon was observed after a third injection when the cooled stomach was rewarmed. However, no adverse effect of gastric cooling on hepatic BSP clearance in normal subjects was directly demonstrated.

Serum amylase activity, blood urea nitrogen concentration and hematocrit were not affected by gastric cooling.

The authors wish to thank Miss Sigrid Funk for her technical assistance and Mrs. Eunice Petrychko for typing the manuscript.

References

1. Turner, G. R. *et al.*: Local gastric hypothermia in management of massive upper gastrointestinal hemorrhage, *Surgery*, **53:** 609, 1963.
2. Nicoloff, D. M. *et al.*: Local gastric hypothermia in the management of massive gastrointestinal hemorrhage, *Surg. Gynec. Obstet.*, **114:** 495, 1962.
3. Sherlock, S.: Diseases of the liver and biliary system, 3rd ed., Blackwell Scientific Publications, Oxford, 1963, p. 200.
4. Wangensteen, S. L. *et al.*: Physiologic effects of gastric hypothermia. I. The effect on estimated hepatic blood flow in the normal anesthetized dog, *Surgery*, **50:** 651, 1961.
5. Orahood, R. C., Wangensteen, S. L. and Luke, W. K.: Physiologic effects of gastric hypothermia. II. The effect on renal blood flow, renal plasma flow, and glomerular filtration rate in the normal anesthetized dog, *Surgery*, **50:** 656, 1961.
6. Chambers, W. N. and Moister, F. C.: Bromsulfalein reaction, *Amer. J. Med.*, **5:** 308, 1948.
7. Morey, G., Gabuzda, G. J. and Scudamore, H. H.: Sensitization to bromsulfalein (phenoltetrabromphthalein - disodium sulfonate), *Gastroenterology*, **13:** 246, 1949.
8. Mendeloff, A. I. *et al.*: Studies with bromsulfalein. II. Factors altering its disappearance from the blood after a single intravenous injection, *Gastroenterology*, **13:** 222, 1949.

Résumé

Le refroidissement gastrique qui est utilisé maintenant en vue de contrôler les hémorragies du tractus digestif semble ne pas être totalement sans dangers; les auteurs croient, par expériences personnelles, qu'une réduction de la fonction hépatique en relation avec ce traitement est probable. Il est sans doute bien connu que l'hémorragie massive entraîne par elle-même une perturbation de la fonction hépatique, et c'est pourquoi il est malaisé de délimiter avec précision le rôle du re-

froidissement dans ce cas. On a procédé à une étude clinique systématique, portant sur 18 sujets ne présentant rien d'anormal au point de vue hépatique; les fonctions rénales et pancréatiques furent aussi contrôlées. L'excrétion du foie fut mesurée par le test à la bromsulphaleine; de plus, l'amylase sérique, l'azote sanguin et l'hématocrite furent aussi vérifiés. Les sujets furent divisés en deux groupes: un premier groupe de neuf servirent de contrôles; les neuf autres furent soumis à l'introduction d'un ballon gastrique maintenu en place pendant trois heures; le refroidissement fut effectué seulement pendant la deuxième heure. Les résultats montrent une élévation du niveau de bromsulphaleine dans le sang lors du refroidissement; cependant, la fonction se rétablit à la normale lors du réchauffement, lequel semble avoir un effet bienfaisant sur le parenchyme hépatique. Il n'a donc pas été possible de démontrer une action directe du refroidissement sur la fonction hépatique. Les autres épreuves restèrent normales.

MALIGNANT PERICARDIAL EFFUSION*

Review of Hospital Experience and Report of a Case Successfully Treated by Talc Poudrage

B. S. GOLDMAN, M.D., B.Sc.(Med.) and F. G. PEARSON, M.D., F.R.C.S.[C],
Toronto, Ont.

Pericardial effusion is an uncommon complication of metastatic malignancy. However, its occurrence may result in rapidly fatal cardiac tamponade in a patient whose life might otherwise be usefully prolonged by palliative measures. The present communication presents the experience with malignant pericardial effusions at the Toronto General Hospital from 1945 to 1964 and describes a case successfully managed by aggressive surgical measures.

Pathological Aspects

Primary malignancy of the pericardium is extremely rare. The frequency of cardiac metastases in autopsy series is variously reported as 2% to 21%.[2-4, 6, 7, 9, 10] Occasionally, the pericardium is the sole site of metastasis, but it is usually involved as part of a general dissemination.

Carcinoma of the bronchus and carcinoma of the breast are the responsible primary tumours in 21% to 50% of cases.[2] Lymphomas, leukemia and malignant melanoma account for the majority of the remaining cases.[10]

The pericardium may be involved by direct intrathoracic extension, by invasion of systemic or pulmonic veins, or by retrograde lymphatic spread from mediastinal nodes.[3] Bronchogenic carcinoma commonly invades the pericardium by direct extension. Carcinoma of the breast, however, more commonly affects the pericardium by a lymphangitic spread resulting in diffuse lymphatic obstruction and massive effusion. The effusion may be serous, serofibrinous, serosanguineous, frankly bloody, or even purulent. The heart itself may also be the site of metastatic malignancy, but involvement of the pericardium is more common and more extensive.[3, 4, 6, 9]

Clinical Aspects

The diagnosis of clinically significant malignant pericardial effusion may be exceedingly difficult. The resultant syndrome is due to the effect of compression and tamponade, interfering with cardiac filling and forward flow. Jugular venous distension, more prominent with inspiration, may precede severe dyspnea and intractable congestive failure, refractory to ordinary therapy. The lowered cardiac output results in hypotension, particularly in the upright position. Pulsus paradoxus has been a constant and important finding in our experience. The individual physician's interpreta-

*From the Department of Surgery, Toronto General Hospital, and the University of Toronto.

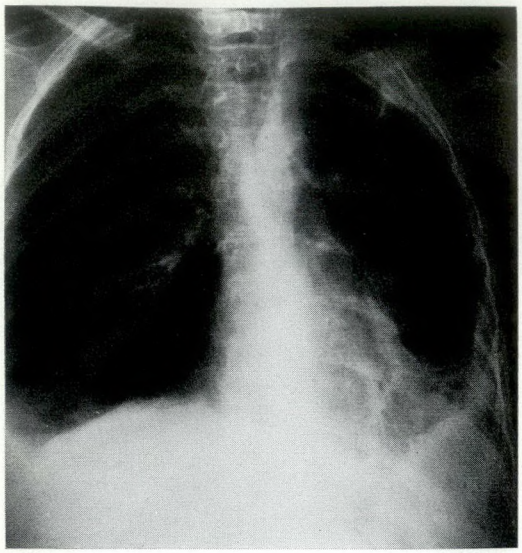


Fig. 1.—Posteroanterior chest radiograph, April 8, 1963. The left pleural space has been successfully obliterated by talc poudrage. The right pleural effusion had been managed subsequently by intrapleural nitrogen mustard and suction drainage.

tion of a diminished intensity of the heart sounds is considered unreliable.

The electrocardiogram is usually nonspecific, presenting changes in voltage and T-waves suggestive only of pericarditis. Chest films may reveal simple cardiomegaly, a globular heart shadow or an irregular nodular contour of the cardiac chambers; the latter may only become obvious on repeated examination of serial plain films. Fluoroscopy may demonstrate a nonpulsating heart border.

The certain diagnosis of pericardial effusion and of its malignant etiology can only be made by needle aspiration through the subxiphoid or parasternal routes, with the demonstration of tumour cells in the aspirate. A little air instilled in the pericardial space after aspiration may demonstrate an irregular cardiac or pericardial contour on a subsequent plain chest film. If the diagnosis is still in doubt after these maneuvers, cardiac catheterization, angiocardiography or open biopsy may be indicated.[12]

Treatment

Where tamponade exists, aspiration of the effusion results in immediate amelioration of distress. Malignant effusions tend to reform rapidly and may require more definitive therapy than repeated pericardiocentesis alone. This may include the intrapericardial instillation of chemotherapeutic agents or colloidal gold, external radiation, or the establishment of adequate drainage by the creation of a pleuropericardial window.[1, 8, 11] The following case report illustrates the effective use of talc poudrage for obliteration of the pericardial space in a patient with a rapidly recurrent malignant effusion and cardiac tamponade.

Case Report

A 44-year-old woman was first admitted to the Toronto General Hospital on November 27, 1962, with a massive left pleural effusion. A diagnosis of metastatic breast carcinoma was established both from pleural biopsy (Abrams' needle biopsy) and cytological examination of the pleural fluid. Primary carcinoma of the left breast had been treated in this patient by radical mastectomy and postoperative radiotherapy 5½ years previously.

On December 5, 1962, an oophorectomy was performed. The left pleural effusion was treated with intrapleural triethylenethiophosphoramide (Thio-Tepa) followed by three days of suction drainage with an intercostal catheter. One month later the left pleural effusion recurred massively and on this occasion was treated by talc poudrage of the left pleural space followed by five days of intercostal-catheter suction drainage. The left pleural space was successfully obliterated and the effusion has not recurred.

Within two months (March 1963) a massive right pleural effusion developed. Again malignant cells were identified in the pleural fluid. On this occasion 20 mg. of nitrogen mustard were introduced into the pleural space followed by four days of suction drainage. At the same time, the patient was given 20 mg. of nitrogen mustard intravenously and was started on oral prednisone. Fig. 1 shows a chest radiograph at the time of discharge.

Within a further six months the right pleural effusion had recurred massively and the patient was readmitted to hospital on September 21, 1963. At this time, she was complaining of extreme dyspnea, throbbing headache and ankle swelling. On examination she had a cushingoid appearance with a moon face, jugular venous distension, hepatomegaly, pitting edema of the lower legs and feet, and the physical signs of a large right pleural effusion (Fig. 2). The cardiac failure and fluid reten-

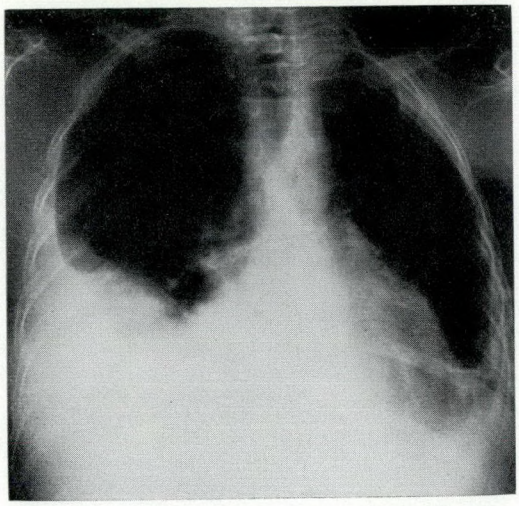


Fig. 2.—Posteroanterior chest radiograph, September 23, 1963. A massive pleural effusion has recurred on the right side, and somewhat obscures the cardiac outline.

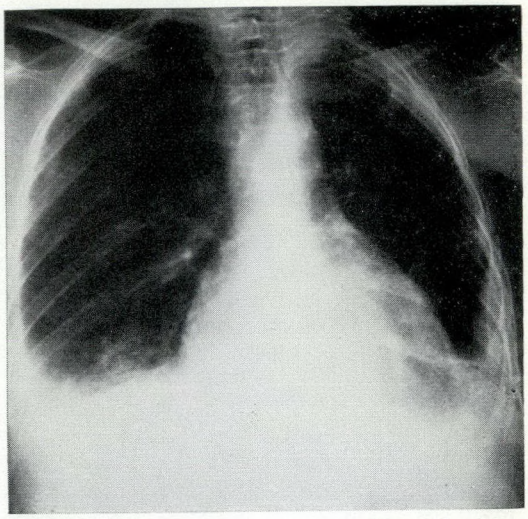


Fig. 3.—Posteroanterior chest radiograph, September 24, 1963. Following aspiration of the right pleural effusion, an enlarged cardiac silhouette compatible with pericardial effusion is apparent, particularly in comparison with Fig. 1. This, however, was not recognized until after diagnosis by needle aspiration of the pericardial space.

tion were initially attributed to the prednisone therapy. The effusion was aspirated, the prednisone was discontinued, and therapy for cardiac failure including diuretics was begun. None the less, her condition continued to deteriorate during the first week after admission. Extreme dyspnea persisted in spite of aspiration of the right pleural effusion and she developed progressive hypotension and a paradoxical pulse. On September 29, 1963, she lost consciousness whenever she attempted to sit up in bed. By September 30 she was more severely hypotensive and semiconscious and her condition was considered terminal. At this time, the possibility of pericardial effusion with tamponade was considered and 300 c.c. of grossly bloody fluid were aspirated from the pericardial space. This was followed by immediate and dramatic improvement with a return of normal blood pressure, a full return of consciousness and relief of dyspnea. Malignant cells were identified in the pericardial fluid. Only in retrospect was the enlarged cardiac shadow recognized in the chest radiograph (Fig. 3).

It was our opinion that talc poudrage offered the best prospect for control of the recurrent right pleural effusion and it appeared reasonable to employ the same technique for the management of the pericardial effusion. On October 2, 1963, the right pleural space and pericardium were explored through a small right anterior thoracotomy incision. Immediately after induction of general anesthesia and positive pressure respiration, the patient became grossly hypotensive, a state that was relieved by the aspiration of a further 200 c.c. of bloody pericardial fluid. Her blood pressure and circulation remained stable throughout the remainder of the operation. At thoracotomy the parietal and visceral surfaces of both the right pleural and pericardial spaces were diffusely involved with thin reticulated strands and plaques of tumour. No bulky deposits of tumour were seen in any area, emphasizing the fact that the patient's disability arose from the mechanical effects of fluid accumulation rather than from the direct effects of gross tumour deposits. Sterile talc was dusted over the surfaces of the pericardial and right pleural spaces; a moderate segment of pericardium was resected from the right side to allow initial communication of pericardial and right pleural spaces, and the pleural cavity was drained. Suction was maintained for four days.

The patient has since developed osseous metastases and has undergone bilateral adrenalectomy (November 20, 1963). There has been an excellent objective response to adrenalectomy, well sustained up to the time of last follow-up examination in September 1964. There has been no recurrence of the pericardial effusion or of either pleural effusion (Fig. 4), and she remains sufficiently well to lead an active and comfortable life. It is now one year since the operation was performed to obliterate the pericardial space.

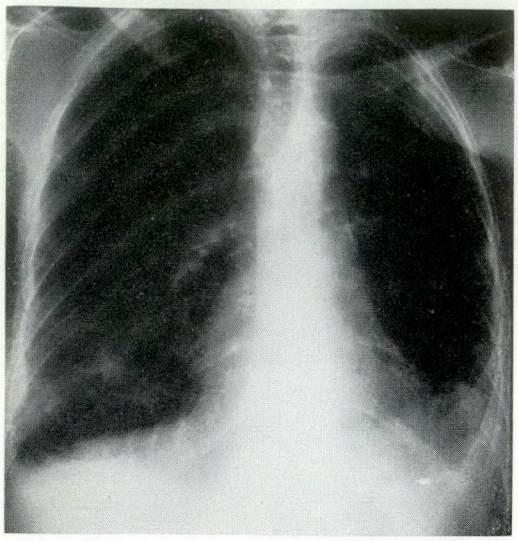


Fig. 4.—Posteroanterior chest radiograph, June 4, 1964. There is no recurrence of fluid in the pericardial space or in either pleural space nine months from the time of right thoracotomy and talc poudrage.

Hospital Experience

For the 19-year period between 1945 and 1964, the records of the Toronto General Hospital contain only 15 cases of malignant pericardial effusion. It is our impression that more cases would have been available for study had the system of indexing included all the complications and sequelae of the primary malignancy.

Only two of the 15 patients were discharged from hospital alive, including the patient described in this report. In 11 cases, pericardial effusion, with or without tamponade, was suspected before death and in the remaining four, the diagnosis was made at autopsy.

Of those who died, significant interference with cardiac action from mechanical compression was considered as either the primary cause or a major contributing factor in 10 of the 13. In four of the patients, cardiac tamponade was the first indication of malignancy.

The primary tumours are listed in Table I. The incidence is similar to other reports.[2, 4] The table emphasizes the fact that almost half (7 of 15) of our patients had malignancies that were compatible with prolonged survival with modern palliative therapy.

TABLE I.—Site of Primary Tumour in 15 Patients with Malignant Pericardial Effusions

Carcinoma of bronchus	6
Carcinoma of breast	3
Malignant lymphoma	3
Malignant melanoma	1
Osteogenic sarcoma	1
Carcinoma of stomach	1

The treatment employed in the management of these patients was as follows: treatment for cardiac failure only, three patients (all dead); repeated pericardial aspirations, six patients (all dead); intrapericardial chemotherapy, one patient (dead); mediastinal and pericardial radiotherapy, four patients (one alive), and operation, three patients (one alive).

Discussion

Palliative therapy has a definite role in the management of many patients with incurable metastatic carcinoma. The survival time in such patients has been significantly prolonged and suffering alleviated by the advent of the newer chemotherapeutic agents, a better understanding of the influence of hormonal environment, and improved techniques of radiotherapy. Although the incidence of pericardial metastases is not high, the development of pericardial effusion may be an important local complication in disseminated malignant disease. Thurber, Edwards and Achor[10] stated that malignant pericardial effusion produced significant interference with cardiac action in 86% of their patients with metastatic involvement of the pericardium.

There is a definite tendency to consider malignant pericardial effusion as a hopeless terminal event. From our own small experience, it is apparent that in a proportion of such patients, active treatment may provide useful palliation with relief of the cardiac tamponade and prolonged survival. This is particularly true in those patients in whom the primary malignancy is in the breast, or is a lymphoma. It is obvious that no program of management can be applied when the diagnosis is only made at autopsy. It cannot be overemphasized that one must first consider the possibility that effusion and tamponade exist, and then carry out needle aspiration as soon as the suspicion is aroused. Despite the presence of known

metastases, adequate treatment may on occasion require a major surgical operation.

Summary

Experience at the Toronto General Hospital with 15 cases of malignant pericardial effusion over the past 19 years is presented and the literature reviewed briefly. It is significant that cardiac tamponade was the main immediate cause of death in 10 of our 15 patients.

One patient is reported in detail in whom malignant pericardial effusion was successfully controlled with talc poudrage of the pericardial space. This method of treatment has not been reported previously.

It is concluded that a proportion of patients with clinically important malignant pericardial effusion may obtain useful palliation and prolonged survival by relief of the effusion and tamponade. This is particularly true when the primary malignancy is in the breast, or is a lymphoma.

Methods of treatment of malignant pericardial effusion are outlined and an aggressive surgical approach is recommended in selected cases.

References

1. Abrahamsen, A. M. and Broch, O. J.: Chronic exudative pericarditis treated by pleuro-pericardial fenestration, *Acta Med. Scand.*, **172:** 359, 1962.
2. Bisel, H. F., Wroblewski, F. and LaDue, J. S.: Incidence and clinical manifestations of cardiac metastases, *J. A. M. A.*, **153:** 712, 1953.
3. Cohen, G. U., Peery, T. M. and Evans, J. M.: Neoplastic invasion of the heart and pericardium, *Ann. Intern. Med.*, **42:** 1238, 1955.
4. DeLoach, J. F. and Haynes, J. W.: Secondary tumors of the heart and pericardium; review of subject and report of 137 cases, *A.M.A. Arch. Intern. Med.*, **91:** 224, 1953.
5. Dimmette, R. M.: The ante mortem diagnosis of secondary tumors of the heart; report of four cases, *U.S. Armed Forces Med. J.*, **1:** 750, 1950.
6. Genovese, P. D. and Fouts, D. B., Sr.: Metastatic tumours of the pericardium, *J. Indiana Med. Ass.*, **53:** 267, 1960.
7. Lamberta, F., Nareff, M. J. and Schwab, J.: Metastatic carcinoma of the pericardium, *Dis. Chest*, **19:** 528, 1951.
8. Mannix, E. P., Jr. and Dennis, C.: The surgical treatment of chronic pericardial effusion and cardiac tamponade, *J. Thorac. Surg.*, **29:** 381, 1955.
9. Scott, R. W. and Garvin, C. F.: Tumors of the heart and pericardium, *Amer. Heart J.*, **17:** 431, 1939.
10. Thurber, D. L., Edwards, J. E. and Achor, R. W. P.: Secondary malignant tumors of the pericardium, *Circulation*, **26:** 228, 1962.
11. Williams, C. and Soutter, L.: Pericardial tamponade; diagnosis and treatment, *A.M.A. Arch. Intern. Med.*, **94:** 571, 1954.
12. Wood, P. H.: Diseases of the heart and circulation, 2nd ed., Eyre and Spottiswoode, Ltd., London, 1956.

Résumé

L'exsudation de liquide cancéreux est une complication qui frappe rarement le péricarde, mais qui est grave, car elle peut entraîner une tamponnade du myocarde. C'est ainsi qu'un malade peut mourir, alors que son état général pourrait permettre une plus longue survie. Les auteurs résument ici l'histoire d'un cas. C'est celui d'une femme âgée de 44 ans, admise au "Toronto General Hospital" en novembre 1962 pour une importante effusion pleurale gauche. Par examen microscopique, on établit le diagnostic: pleurésie métastatique ayant son origine dans un carcinome du sein; la patiente avait subi une mastectomie cinq ans auparavant. En décembre, on effectue une ovariectomie, et l'exsudation pleurale est traitée par l'administration locale de Thio-Tepa, suivie d'aspiration continue. Un mois plus tard, il est procédé à un poudrage au talc de la cavité pleurale gauche, qui s'oblitère. Cependant, en mars 1963, les mêmes troubles se reproduisent à droite, traités par application de moutarde. La patiente est rehospitalisée en septembre pour une récidive; l'état général est beaucoup plus mauvais: il existe une enflure importante de la face et une forte dyspnée. L'exsudation pleurale est aspirée, et l'on découvre une péricardite cancéreuse, confirmée par l'examen microscopique. On pratique un poudrage au talc des cavités pleurale droite et péricardique. L'amélioration fut très grande et la malade put reprendre une certaine activité. Dans les archives de l'hôpital, il fut possible de retrouver 15 cas de ce genre traités pendant les 19 dernières années: dans 10 de ces cas, la mort survint par suite d'une tamponnade du cœur. Les auteurs insistent sur le fait que, bien que l'exsudation cancéreuse du péricarde soit considérée comme sans espoir, il y a lieu de lutter la tamponnade qui est la raison du décès. Un traitement symptomatique est fortement indiqué.

THE OBSTRUCTIVE FACTOR IN URETHRAL DIVERTICULUM IN THE PARAPLEGIC PATIENT*

J. G. CONNOLLY, M.D., F.R.C.S.(Edin.), F.R.C.S.[C] and
A. W. BRUCE, M.B., Ch.B., F.R.C.S., F.R.C.S.(Edin.), F.R.C.S.[C], *Kingston, Ont.*

BEFORE 1950 there were few reports in the medical literature of urethral diverticula occurring in the male. Many of those recorded were associated with urethral stricture or injury, for example, those of Ostry[1] and McKay and Colston.[2] About this time a series of papers were published describing the urethral complications in paraplegics on prolonged catheter drainage; these included periurethral abscesses, fistulas and diverticula. All of the patients with diverticula had had varying periods of urethral catheter drainage and the mechanism of the production of the diverticula was felt to be well understood. Prolonged catheter drainage resulted in injury to the urethral wall and the production of a pressure sore. If this damage was extensive and destruction of the urethral wall occurred, a diverticulum resulted.

In most cases there was no evidence that obstruction played an important role and in Pate and Bunts' well-documented series[3] of 28 cases, only one case was noted in which the obstructive element was thought to be important. This, however, has not been our experience as the following cases illustrate.

CASE 1.—Mr. J.M. developed a neurogenic bladder after fracture dislocation of the twelfth thoracic vertebra. This condition was treated initially by Foley catheter drainage. Six months later the patient developed a periurethral abscess that was followed by fistula formation. The fistula was closed surgically. One year later there was no evidence of a diverticulum. However, after cessation of catheter drainage, a penile clamp was employed to control incontinence and at the end of a further year, a slight swelling was noted on the ventral surface of the penis. The recent urethrogram demonstrates a well-marked diverticulum (Fig. 1).

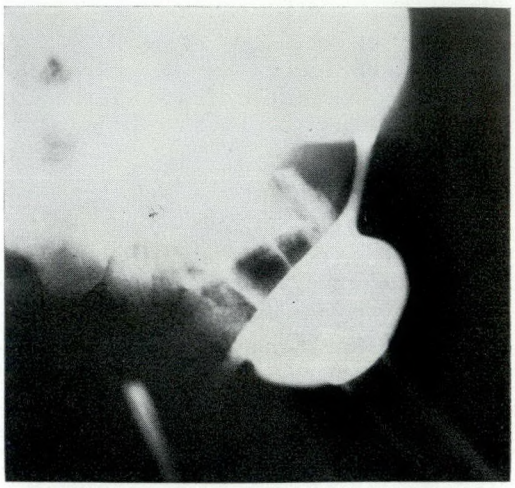


Fig. 1.—Urethrogram showing large diverticulum in penile urethra.

CASE 2.—Mr. H.R. developed a neurogenic bladder as a result of prolapsed intervertebral discs. For a period of several months he was treated with an indwelling Foley catheter with no apparent urethral reaction. A penile clamp was then used to control incontinence and six months later a soft swelling was noted on the ventral surface of the penis. Urethrography demonstrated a large diverticulum (Fig. 2).

CASE 3.—Mr. R.R. developed paraplegia and a neurogenic bladder following a gunshot wound of the lower thoracic vertebrae. Initial treatment consisted of Foley catheter drainage followed later by condom drainage. In attaching the condom a tight band of Elastoplast was placed just proximal to the glans and within a year a soft fluctuant swelling was noted on the undersurface of the penis (Fig. 3). This presence of a diverticulum was confirmed by urethrography (Fig. 4).

DISCUSSION

The incidence of urethral diverticula in paraplegics is difficult to determine. Comarr and Bors[4] investigated 619 such patients and found 19 diverticula. In performing

*Department of Urology, Queen's University, Kingston, Ont.

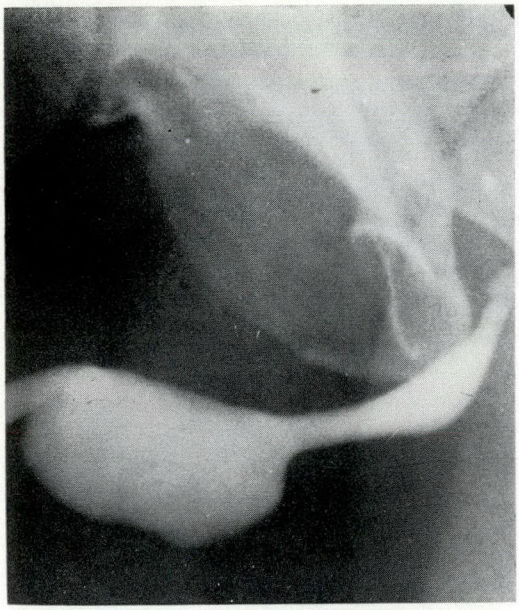


Fig. 2.—Large fusiform diverticulum in anterior urethra as shown by retrograde urethrogram.

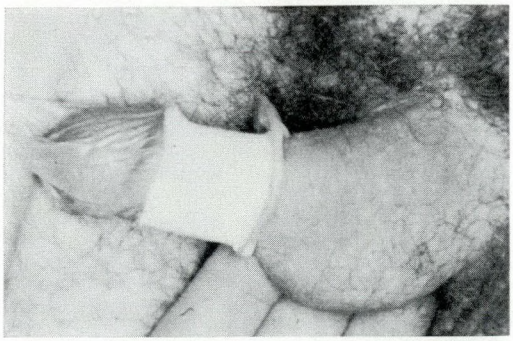


Fig. 3.—Diverticulum showing its relationship to the constricting ring of Elastoplast used for attaching condom drainage.

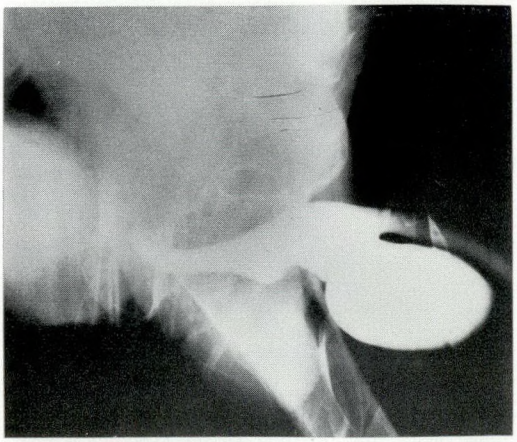


Fig. 4.—Urethrogram showing diverticulum demonstrated in Fig. 3.

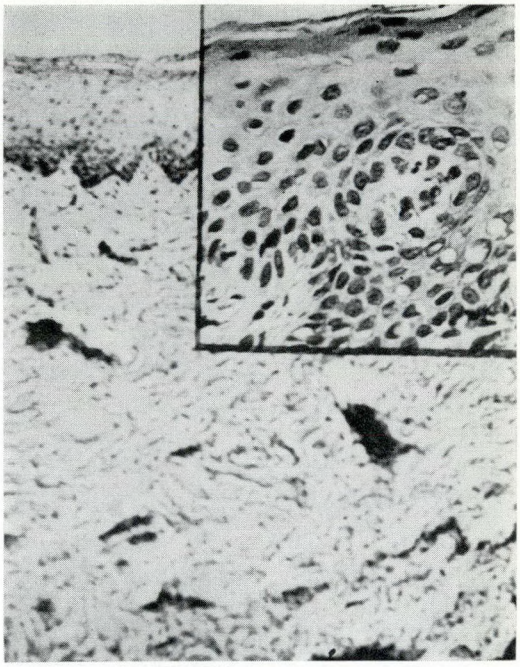


Fig. 5.—Section of urethral diverticulum showing thickened epithelial surface, and fibrosis in wall of sac. Inset shows in higher power the epithelial surface which is obviously squamous in type.

urethrograms on 122 paraplegics, they demonstrated 61 diverticula. However, many of these were 1 cm. or less in diameter and should not, perhaps, be accepted as evidence of pathological change. Griffiths and Walsh,[5] on the other hand, found 19 diverticula in 200 paraplegics investigated by urethrography and Bunts[6] reported an incidence of 4.6% in 1000 paraplegics. It would seem that these lower figures are more realistic and represent the true incidence.

In each case injury to the urethral wall was associated with the insertion of an indwelling urethral catheter. With extensive injury and formation of a large defect, diverticulum formation results. With less destruction or where some degree of healing occurs, this need not happen. In these cases, provided there is free drainage of urine, diverticulum formation may be avoided. However, if there is distal obstruction, a diverticulum may develop at the weakest point in the urethra. It is this set of circumstances that produces the delayed type of diverticulum in paraplegics.

A small diverticulum may be asymptomatic. If it is of moderate size and situated in the anterior urethra, the diverticulum may be palpable when filled and can usually be emptied manually. The urinary symptoms associated with urethral diverticulum may include frequency, dysuria, discharge and dribbling after micturition. The complications include inflammation, periurethral abscess, urethrocutaneous fistula and calculi.

The pathological findings are quite constant. The epithelial lining usually undergoes squamous metaplasia. The epithelial glands are not seen and deep to the mucosa there is a fibrotic replacement of normal tissue. There may or may not be round-cell infiltration (Fig. 5).

The prophylaxis of this condition is based upon techniques which will minimize urethral reaction. The use of small siliconized and sanitized Foley catheters has greatly reduced the incidence of urethritis.[7] Ross, Gibbon and Damanski[8] have also shown the advantage of using small plastic catheters for bladder drainage in paraplegics.

In the management of the post-catheter phase, obstruction to the urethral lumen must be avoided. Penile clamps and tight bands applied for the attachment of various drainage appliances are the most common causes. If penile clamps are used to control incontinence, they must be carefully supervised and repeated urethrograms should be performed. If any dilatation of the urethra is demonstrated, condom or bag drainage should be employed. It is important to support the urethra with Elastoplast strapping when condom drainage is instituted. An alternative procedure is to attach the drainage apparatus to the anterior abdominal wall. With either method the urethra is spared the effects of a constricting ring.

The management of a urethral diverticulum may be conservative or operative. It is best to treat small diverticula conservatively. This includes eradication of urinary tract infection, elimination of obstruction and manual emptying of the diverticulum. The main indication for operative removal is urethral obstruction due to compression, a feature of very large diverticula. The other indications for operative intervention include recurrent or persistent infection, calculus and fistula formation. The operative treatment consists of removing the sac of the diverticulum and closing the urethral defect. If the urethral opening is small, it may be occluded by a purse-string suture of fine chromic material. The suture line must be reinforced by as many layers of tissue as can be obtained. Larger urethral openings can be managed by marsupialization. When urethral healing has occurred, continuity of the urethra is restored using the Denis Browne procedure or Cecil's urethroplasty.

Summary

Three paraplegic patients with urethral diverticula are presented. These patients illustrate the importance of the obstructive factor in the development of the delayed type of urethral diverticulum.

References

1. Ostry, H.: Diverticulum of urethra in a male, *Canad. Med. Ass. J.*, **58:** 68, 1948.
2. McKay, R. W. and Colston, J. A. C.: Diverticula of male urethra; report of 10 cases, *Surg. Gynec. Obstet.*, **48:** 51, 1929.
3. Pate, V. A., Jr. and Bunts, R. C.: Urethral diverticula in paraplegics, *J. Urol.*, **65:** 108, 1951.
4. Comarr, A. E. and Bors, E.: Pathological changes in urethra of paraplegic patients, *J. Urol.*, **66:** 355, 1951.
5. Griffiths, I. H. and Walsh, J. J.: Diverticula and fistulae of the urethra in paraplegics, *Brit. J. Urol.*, **33:** 374, 1961.
6. Bunts, R. C.: Management of urological complications in 1000 paraplegics, *J. Urol.*, **79:** 733, 1958.
7. Bruce, A. W. and Quirk, J.: Prostatectomy and infection, *J. Urol.*, **92:** 523, 1964.
8. Ross, J. C., Gibbon, N. O. K. and Damanski, M.: Recent developments in the treatment of paraplegic bladder, *Lancet*, **2:** 520, 1957.

Résumé

Avant 1950, on ne trouve dans la littérature médicale que peu de choses au sujet des diverticules urétraux chez l'homme; d'une façon générale, ces cas étaient associés aux blessures ou à des traumatismes divers. Plus récemment, un certain nombre d'articles est paru, concernant des diverticules secondaires à des troubles d'innervation de la région: c'est le cas des paraplégiques ayant subi une cathétérisation prolongée. Les auteurs résument ici l'histoire de trois malades. Chacun d'eux était atteint d'une lésion de la

moelle épinière avec paralysie vésicale. Ceci avait donc nécessité un drainage de longue durée par cathéter et une constriction du pénis en vue d'assurer l'étanchéité. Ces trois cas développèrent des diverticules de l'urètre, demontrés par l'urétrogramme. L'incidence de cette complication est difficile à déterminer; elle varie beaucoup selon les auteurs. Du point de vue symptomatologique, un diverticule de petite taille peut être parfaitement inaperçu; lorsqu'ils sont de taille plus grande, ils peuvent former une petite masse palpable le long du pénis; ils provoquent de la dysurie, et de la pollakiurie. Les complications en sont l'inflammation, les abcès periurétraux, les fistules à la peau et les calculs. L'histologie montre de façon constante une métaplasie de l'épithelium, celui-ci devenant cornifié. La prophylaxie de cette affection consistera à utiliser des cathéters de petite taille, siliconés, et à éviter les pinces péniennes parfois employées pour lutter contre l'incontinence. Le traitement des petits diverticules doit être conservatif. Les plus gros, compliqués de calculs ou provoquant des infections seront justiciables de l'intervention. Celle-ci consiste en l'excision du sac diverticulaire et suture des tissus en plans aussi nombreux que possible.

THERMAL BURN ENCEPHALOPATHY

W. K. LINDSAY. M.D., F.R.C.S.[C],* E. G. MURPHY, M.B., B.S.(Lond.), D.C.H.(Eng.)† and D. C. BIRDSELL, M.D.,‡ *Toronto, Ont.*

THE neurological effects of burns is not well understood. In this communication, a short review of the subject will be presented, as well as a report of two cases of encephalopathy following thermal burns. It may or may not be significant that these two patients received high doses of the polypeptide antibiotic, colistin.

Historically, little has been written about the pathological changes in the nervous system following burns and even fewer conclusions can be drawn from what has been written. Riehl, in 1931, recognized that people dying in the period of shock following severe burns had cerebral edema.[3] He postulated that many of the clinically observed phenomena were based on this edema.

Erb, Morgan and Farmer[2] reported finding cerebral vascular congestion and edema at autopsy in patients with fatal burns. In one instance petechial hemorrhages were found in the floor of the fourth ventricle.

According to Sevitt,[3] the presence of cerebral edema following fatal burns has been confirmed by many workers. Edema was attributed to anoxia owing to circulatory disturbance. Sevitt raises a reasonable objection to this explanation saying that it is doubtful whether anoxia alone ever produces edema of the brain or any other tissue. He also points out that Wilson *et al.* in 1938 suggested that the altered mental state, fever, vomiting, circulatory disturbances and even death were the result of the release of a burn toxin.[3]

The presence of a burn toxin is still a controversial subject and is of experimental interest. No evidence has ever been presented to show that burned areas release any substance which produces neuropathology. The role of burns in the production of cerebral edema, cerebral vascular changes, or other forms of pathology in the nervous system is still under investigation.

CLASSIFICATION OF NEUROLOGICAL EFFECTS OF BURNS

The classification of neurological effects of burns shown in Table I is suggested.

A. *Extracranial Effects*

The effects of surface burns on the spinal cord and autonomic nervous system are unknown. Individual cells in these locations would probably be affected like other cells; that is, there is a spectrum of change varying with the intensity and duration of

*Associate Surgeon, The Hospital for Sick Children, Toronto; Assistant Professor of Surgery, University of Toronto.

†Assistant Physician, The Hospital for Sick Children, Toronto; Clinical Teacher, Department of Medicine, University of Toronto.

‡Fellow, The Research Institute, The Hospital for Sick Children, Toronto.

TABLE I.—Classification of Neurological Effects of Burns

A. *Extracranial effects*
   1. Spinal cord
   2. Autonomic nervous system
   3. Peripheral nerves

B. *Intracranial or cerebral effects*
   1. *Early*
      (a) Related to direct heat to the cranium
      (b) Related to shock
   2. *Late*
      (a) Encephalopathy
      (b) Psychological disturbance
      (c) Meningitis
      (d) Secondary, e.g., uremia

exposure to the heat source. This ranges from reversible nuclear and cytoplasmic swelling to coagulative necrosis. The autonomic nervous system regulates capillary dilatation in burned areas.[4] Extensive burning blocks this vascular response in the burned area, probably because of local destruction. Central nervous system changes have not been investigated. It is unlikely that permanent central damage results because burn-scar capillaries have the ability to contract and dilate. The effects of surface burns on peripheral nerves are almost as poorly understood. A fact of practical importance is that nerve endings for pain in the skin are preserved if only partial skin loss has been suffered. Sensation is lost if full-thickness skin loss has occurred. Muscle spasm and gross tremor are common observations in patients who have been recently burned. Carey *et al.*[5] offered an explanation for this suggesting that there was an accentuation and aggravation of motor-end plate activity. Many observers attribute this spasm and tremor to fluid and electrolyte imbalance.

B. *Intracranial Effects*

1. *Early effects.*—(a) Early cerebral changes may be due to brain damage caused by the heat from a scalp burn. Burns of sufficient severity to affect the brain directly are usually fatal.

(b) Other early cerebral changes probably have a complex origin, and may be related to one, or a combination, of shock, hypoxia or carbon-monoxide poisoning. Little has been reported concerning cerebral changes during the phase of neurogenic shock. Death during this phase is due primarily to obstruction of the respiratory passages.[6] During the next phase of burn shock, that is, the period of fluid and electrolyte disturbance, cerebral effects have been noted clinically and at autopsy. Emery and Reid[7] reported six cases of minor burns in children which resulted in convulsions within the first 48 hours. All but one of these children died and the autopsy finding common to all was cerebral edema and herniation of the cerebellum. The living child is now an athetoid spastic.

2. *Late effects.*—(a) The late cerebral effects of burns, which are broadly classified under the term encephalopathy, may be related pathologically to the effects observed by Emery and Reid. The exact relationship between the surface burn and the cerebral changes and the reason for the delay in the manifestations of the cerebral lesion is not known. In one of the cases reported by these workers, convulsions began eight days after relatively minor burn. This child died eventually and at autopsy only cerebral edema was noted.

Emery and Reid[7] reviewed the literature and found only four cases of encephalopathy following burns in children and very few in adults. In all of these cases the onset of cerebral symptoms in the patients was delayed until from 10 to 30 days after the burn. It is conceivable that at this late stage other factors are involved; however they were not obvious.

Some other cases described in their review included: Temporary blindness and mental deficiency beginning 10 days after burning; convulsions and subsequent mental deficiency 26 days after a burn; "cerebral" death six months after burning (the pathology in this last case resembled that of multiple sclerosis and raised the possibility of acute disseminated encephalomyelitis).

Sevitt[3] reports three further cases of burns in children which terminated in "cerebral" deaths at periods of three, five and 58 days after severe surface burns. In the early deaths, small areas of hemorrhagic necrosis were found in the cortex and around the lateral ventricles. There were also necrotic areas and thrombosed

vessels at scattered locations. The late death appeared to be related to a small area of hemorrhagic necrosis in the subcortical region of the parieto-occipital area. The two cases described in the present paper fall into this late group, having developed cerebral symptoms weeks after the burn.

Emery and Reid in their review of the literature mention that mental deficiency was noted in three of the four children who developed encephalopathy following surface burns. It would be difficult to prove that mental deficiency was a specific effect of the burn because the premorbid status is usually not known, and the condition could have resulted from prolonged hypoxia suffered at the time of the burn.

(b) Psychological disturbance is a common and important complication of burns, particularly in children. Here again the premorbid condition is important but not always known. The emotional reaction commonly follows a pattern. Early in the post-burn period, patients may be noisy and demanding. Children have outbursts of rage and irrational behaviour. Later the reaction is frequently one of withdrawal and complete dependency. Underlying all these reactions is the anxiety produced by the accident, the hospital situation, the fear of death, the appearance of the burned areas, and the [illegible] procedures. A period of euphoria may follow this phase as the patient represses the experience and denies his present situation or rationalizes that it all did him some good. These early reactions often disappear as soon as the burns are grafted or epithelialized. The late psychological effects are related to the unpleasant memory of the accident and the readjustment necessary because of the disfigurement and physical handicaps. Patients have a fear of rejection and feelings of guilt and inadequacy. There is good evidence also in burned children that separation from the parents during the prolonged hospital stay is a factor in subsequent psychological disturbance.

(c) The cerebral effects of associated meningitis and brain abscess are included here for completeness. Since virtually every severe burn is infected, the possibility of septicemia and metastatic spread of infection to the brain always exists. Delirium associated with the high temperatures accompanying septicemia is transitory. After the second or third week, septicemia is less common because granulation tissue provides a barrier against the invasion of bacteria into the wounds. Fortunately meningitis and brain abscesses are rare complications. Only one instance of brain abscess was found among 600 cases of burns reviewed at The Hospital for Sick Children (1956-1960).[9]

(d) Cerebral changes in the post-burn period may be secondary to a complication of the burn, for example, uremia following severe renal damage. Another example is hepatic coma following treatment with tannic acid. However, this mode of treatment is obsolete and this complication is no longer seen.

Case Reports

Two children, admitted to The Hospital for Sick Children in 1963 with severe thermal burns, developed convulsions late in the recovery period. A summary of the cases follows:

Case 1.—On July 15, 1963, a healthy 8-year-old boy was playing at a service station when he received 70% to 75% body burns in a gasoline explosion (Fig. 1). Thirty per cent of the burn was judged to be third degree. He was treated in a regional hospital where an intravenous cut-down, tracheotomy and bladder catheterization were carried out. Therapy included transfusion of blood and other fluids, the administration of antibiotics, hydrocortisone (Solu-Cortef) and sedation. Five days after the burn, skin homografts taken from his mother were applied to his thighs and buttock. His temperature remained high and his condition was not good. Twenty days after the burn he was found to have an *E. Coli* septicemia and his wound culture grew *B. pyocyaneus*. At this time he was admitted to The Hospital for Sick Children where it was discovered that he also had bilateral pneumonia. Despite the administration of several antibiotics in high doses, cultures of his blood continued to yield enteric streptococcus and he required repeated blood transfusions. Among other drugs, he received 2100 mg. of colistimethate sodium (Colymycin) in 27 days. Four weeks after the burn, respiratory distress developed which was relieved after tracheal suction.

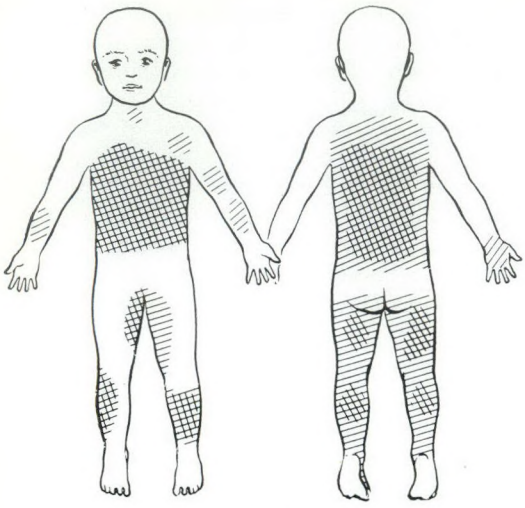


Fig. 1

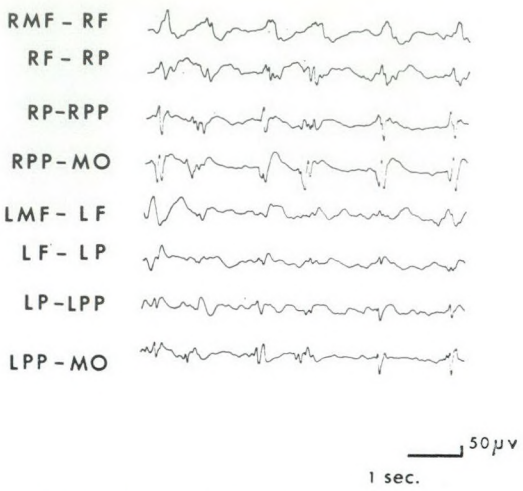


Fig. 3.—Case 1. EEG taken 51 days after injury shows considerable seizure activity, which is particularly prominent in the right head region. The background is very poorly organized.

Thirty-three days after the burn, convulsions began which started on the right side and then became generalized. The seizures were partially controlled with paraldehyde and diphenylhydantoin sodium (Dilantin). He lapsed into a coma from which he never recovered. An electroencephalogram (EEG) taken on the thirty-eighth day was grossly abnormal (Fig. 2) and showed poorly organized slow activity in all head regions at 1-3 and 4-7 cycles per second (c.p.s.). There was no focal factor or seizure activity.

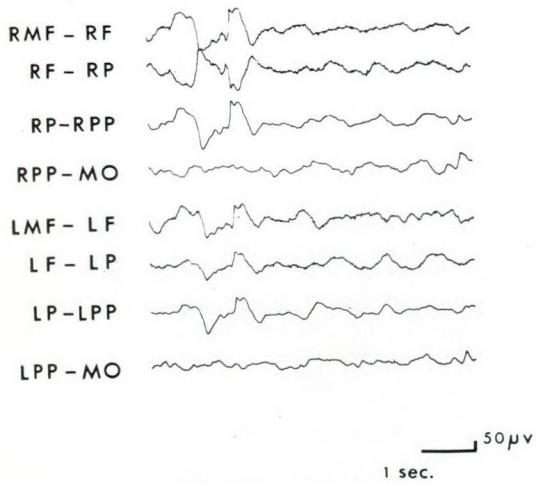


Fig. 2.—Case 1. EEG taken 38 days after the burn shows poorly organized activity in all head regions and is diffusely abnormal.

Clinically he continued to have fixed dilated pupils and nystagmoid movements of the eyes. Left-sided twitching became gradually more prominent. A second EEG taken on the fifty-first day showed considerable seizure activity (Fig. 3). There was high-voltage spike activity associated with slow waves at 1-3 c.p.s. continuous throughout the record. There was some asymmetry with the spikes of a higher voltage in the right head regions.

He died on the fifty-second day. At autopsy he had acute bacterial endocarditis, septic infarction of the spleen, acute meningitis and encephalitis. The pathological examination of the brain revealed congestion of the meninges and cortex. There was thickening and softening of the meninges especially over the cerebral hemispheres. No focal abnormalities, hemorrhages or abscesses were found. Histologically there was acute inflammatory infiltration of the meninges and scattered inflammatory foci in the cortex and white matter. No perivascular hemorrhages were found.

Case 2.—On July 10, 1963, a healthy 12-year-old boy received an 80% body burn in a house fire (Fig. 4). Forty per cent of this burn was judged to be third degree. He was first treated in his regional hospital with transfusions of blood and other fluids, and the administration of antibiotics and steroids. A tracheotomy was carried out and he was catheterized. Five days after the burn, *B. pyocyaneus* and streptococcus were isolated from a blood culture. He was started on colistimethate sodium, 125 mg. three times daily, on the ninth

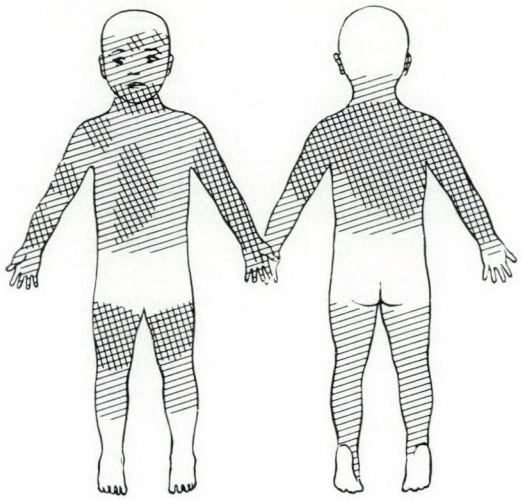


Fig. 4

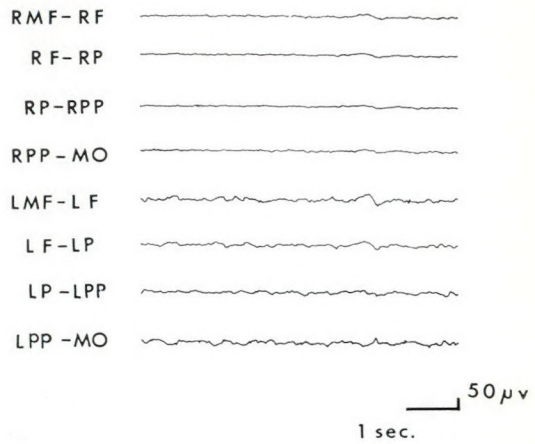


Fig. 5.—Case 2. EEG taken 35 days after the burn shows very little activity in the right hemisphere and poorly organized activity in the left hemisphere.

day after injury. The same day he developed wandering speech and hallucinations. He was admitted to The Hospital for Sick Children on the tenth day. Blood taken six hours after colistimethate sodium was started failed to grow any bacteria. His irrational behaviour improved. On the twenty-eighth day, he was taken to the operating-room for autogenous grafting. On the thirty-second day, he developed convulsions which started on his right side and soon became generalized. These continued to recur for 27 hours despite combined Luminal-Dilantin therapy. He finally returned to a normal level of consciousness and no neurological deficit was noted. Physical examination was limited by his burn dressing. His behaviour, however, was considered abnormal in that he was irritable and unco-operative. The EEG three days after the seizure showed very little cerebral activity of the right hemisphere (Fig. 5). The left side showed poorly organized activity of low to medium voltage of 1-3 and 4-7 c.p.s.

His behaviour and general condition improved. Dilantin in a dose 1 grain twice daily prevented further seizures.

The second EEG on the forty-second day showed a marked improvement; however, the voltage was lower in the right head regions (Fig. 6). The predominant activity was at 8 c.p.s.

Neurological examination on the sixtieth day revealed clawing of the left hand with weakness and wasting of the small muscles. Tendon jerks on the left arm were relatively brisk, including the finger jerk. The lower limb tendon jerks were brisk and equal. There was a positive Babinski response on the left side. This was interpreted as evidence of a pyramidal disturbance involving the left side, which correlated well with the EEG asymmetry.

He was discharged home on the sixty-first day. His only medication was Dilantin, 1 grain, twice daily. This was continued for seven months and then discontinued when he was readmitted to hospital. On follow-up neurological examination, he was intellectually and emotionally normal. Slight weakness and wasting of the small muscles of his left hand were

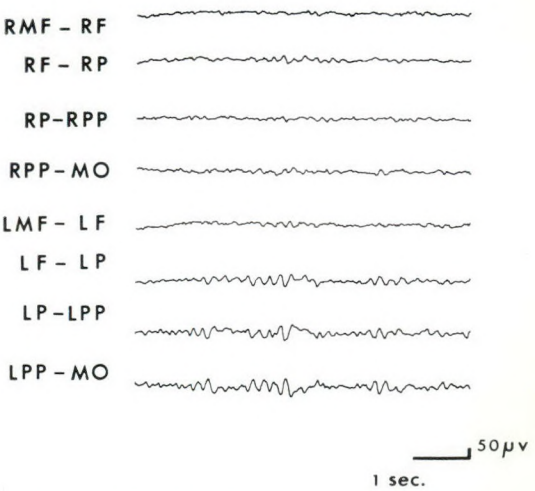


Fig. 6.—Case 2. EEG taken 42 days after thermal injury shows considerable improvement, but there is persistent asymmetry with relatively low voltage in the right hemisphere.

still present. However, a marked improvement had taken place. The Babinski response on the left side was now negative. The EEG was normal. More recent reports indicate that he has been seizure-free.

Related Factors

Gram-negative septicemia, especially that due to *B. pyocyaneus*, has been almost uniformly fatal in the past.[10] Recently a case treated successfully with colistin has been reported.[11] Colistin is a relatively new polypeptide antibiotic isolated by Koyama in Japan in 1950.[12] It is produced by spore-forming soil bacteria and is chemically and clinically similar to the polymyxins. Biserte and Dautrevaus pointed out that colistin and polymyxin B are both pentacyclopeptides which terminate in a hepto-octanoic acid ester.[12] However, colistin contains no phenylalanine and, when given as the methane sulfonate, seems to be less toxic. Others[1, 13-16] have confirmed the similarity to polymyxin and the low toxicity. It is generally agreed that colistin may aggravate pre-existing renal impairment. The only neurotoxicity reported is transient perioral paresthesia[14] and peripheral paresthesia,[13] both of which cleared when the dosage was reduced. All authors agree that colistin is effective against many Gram-negative organisms, especially *B. pyocyaneus*. Organisms commonly resistant to this agent are *E. coli*, *Aerobacter aerogenes*, *Klebsiella pneumoniae* and *Brucella*.

Discussion

These two cases have in common the following features: extensive thermal burns; apparently adequate shock and wound treatment; *B. pyocyaneus* septicemia or wound infection; treatment with high doses of colistin, and neurological disturbance coming on several weeks after the burn, consisting of convulsions, coma and EEG abnormalities. The clinical picture of convulsions followed by coma is extremely rare in the burn population of The Hospital for Sick Children, Toronto. EEG studies have not been necessary in any burn patients previously. Therefore, these two cases were considered unique and worthy of reporting.

Cerebral disturbance in the first case began 19 days before the patient died. At the time of onset of convulsions there were no signs of meningitis. The autopsy findings did reveal acute encephalitis and meningitis. It is therefore difficult to determine the origin of the encephalopathic process.

Cerebral disturbance in the second case was transitory. This patient's EEG study revealed a depression of activity in the right hemisphere and moderate disturbance of function in the left hemisphere. It returned to nearly normal within a week. This suggests the cerebral disturbance was of a moderate degree, which considered with the asymmetrical involvement suggests that the lesion may have been secondary to vascular occlusion. The fact that both of these patients received colistin is worthy of note since this drug is chemically related to known neurotoxins.

Summary

An attempt has been made to classify and review the cerebral effects of surface burns. The cases of two children with severe burns have been reported. Both developed convulsions, coma and abnormal EEG findings, an unusual picture in the burn population at The Hospital for Sick Children, Toronto. The possible nature of this complication is discussed. The fact that both of these patients received very high doses of colistin was felt to be worthy of note.

References

1. Wright, W. W. and Welch, H.: Chemical, biological, and clinical observations on colistin, *Antibiot. Ann.*, **7:** 61, 1960.
2. Erb, I. H., Morgan, E. M. and Farmer, A. W.: The pathology of burns; pathologic picture as revealed at autopsy in series of 61 fatal cases treated at Hospital for Sick Children, Toronto, Canada, *Ann. Surg.*, **117:** 234, 1943.
3. Sevitt, S.: Burns pathology and therapeutic applications, Butterworth & Co. Ltd., London, 1957, p. 276.
4. Sevitt, S.: Discussion: Pathological sequelae of burns, *Proc. Roy. Soc. Med.*, **47:** 225, 1954.
5. Carey, E. J. *et al.*: Studies on ameboid motion and secretion of motor end-plates. VII. Experimental pathology of the secretory mechanism of motor end-plates in thermal shock, *Amer. J. Path.*, **22:** 175, 1946.

6. WARTMAN, W. B.: Mechanisms of death in severe burn injury: the need for planned autopsies, *In*: Research in burns. The Proceedings of the First International Congress on Research in Burns, held at National Naval Medical Center, Bethesda, Maryland, September 19-22, 1960, edited by C. P. Artz, F. A. Davis Company, Philadelphia, 1962, p. 6.
7. EMERY, J. L. AND REID, D. A. C.: Cerebral oedema and spastic hemiplegia following minor burns in young children, *Brit. J. Surg.*, **50:** 53, 1962.
8. WOODWARD, J. M.: Parental visiting of children with burns, *Brit. Med. J.*, **2:** 1656, 1962.
9. SHANDLING, B.: Unpublished data.
10. TUMBUSCH, W. T. *et al.*: The rising incidence of pseudomonas septicemia following burn injury, *In*: Research in burns. The Proceedings of the First International Congress on Research in Burns, held at National Naval Medical Center, Bethesda, Maryland, September 19-22, 1960, edited by C. P. Artz, F. A. Davis Company, Philadelphia, 1962, p. 235.
11. FEKETY, F. R., JR., NORMAN, P. S. AND CLUFF, L. E.: The treatment of Gram-negative bacillary infections with colistin; the toxicity and efficacy of large doses in forty-eight patients, *Ann. Intern. Med.*, **57:** 214, 1962.
12. SCHWARTZ, B. S. *et al.*: Microbiological and pharmacological studies of colistin sulfate and sodium colistinmethanesulfonate, *Antibiot. Ann.*, **7:** 41, 1960.
13. MCCABE, W. R., JACKSON, G. G. AND KOZIJ, V. M.: Clinical and laboratory observations on the use of colistin in infections by Gram-negative bacilli, *Antibiot. Ann.*, **7:** 80, 1960.
14. YOW, E. M. *et al.*: Colistin (Coly-Mycin) in resistant bacterial infections, *A.M.A. Arch. Intern. Med.*, **108:** 664, 1961.
15. CUTHBERT, M. F.: Colistin and polymyxin, *Lancet*, **1:** 383, 1962.
16. BLAUSTEIN, A.: Studies on sodium colistinmethanesulfonate, including a comparative study of its effect on Gram-negative organisms, *Antibiot. Ann.*, **7:** 75, 1960.

RÉSUMÉ

Les auteurs résument ici deux cas d'encéphalopathie survenant comme complication d'une brûlure. Le premier est celui d'un enfant de 8 ans admis d'urgence pour brûlures couvrant 70-75% de la surface du corps; le traitement consista en transfusions, antibiotiques, cortisone et calmants. Cinq jours plus tard, on tente des greffes de peau données par la mère; l'état général reste mauvais et le malade est atteint de septicémie à *E. coli*. Il est alors transféré à l'Hôpital des Enfants Malades de Toronto; il est en outre porteur d'une pneumonie double. Cet état infectieux réagit mal aux antibiotiques. Trente-trois jours après l'accident, l'enfant est soudainement pris de convulsions et l'électro-encéphalogramme montre de grosses anomalies. Le patient entre dans un coma profond dont il ne sortira pas; il meurt le cinquante-deuxième jour après la brûlure. L'autopsie permet de trouver une endocardite bactérienne aiguë, des embolies septiques de la rate, une méningite et une encéphalite aiguë. Du point de vue microscopique, on note une infiltration inflammatoire aiguë des méninges ainsi que des foyers inflammatoires localisés au hasard dans le cortex et la matière grise. Le deuxième cas est celui d'un garçon de 12 ans, atteint de brûlures intéressant 80% de la surface corporelle. Là encore, une septicémie aiguë à streptocoque et bacille pyocyanique s'installe. Des convulsions apparaissent au trente-deuxième jour d'hospitalisation, mais cet état réagit assez bien à la thérapeutique. L'électro-encéphalogramme montre une très faible activité de l'hémisphère droit. Au quarante-deuxième jour l'EEG s'améliore. L'enfant est renvoyé à domicile au soixante et unième jour, sous traitement à la Dilantine, qui est continué pendant sept mois. Une ré-hospitalisation a alors permis de procéder à un examen neurologique complet qui est normal. Les convulsions ont depuis totalement disparu. Les auteurs discutent l'étiologie possible de l'affection. Dans les deux cas présentés ici, il est intéressant de noter que tous les deux reçurent de fortes doses de colistine; cette drogue est chimiquement apparentée à certaines neurotoxines.

REVIEW ARTICLE

SURGERY OF INTRACRANIAL BERRY ANEURYSMS: A REVIEW

K. K. JAIN, F.R.C.S.[C],* *Vancouver, B.C.*

MANAGEMENT of intracranial aneurysms has become one of the major problems in neurosurgery. Agreement concerning definite treatment for these lesions has not been reached by neurosurgeons. The literature contains over 1000 publications relative to this subject including some excellent monographs.[17, 36, 56, 121] This communication presents a review of this literature in which only selected references are given.

CONSERVATIVE VERSUS SURGICAL MANAGEMENT OF RUPTURED BERRY ANEURYSMS

The immediate mortality in subarachnoid hemorrhage from rupture of an aneurysm varies between 50% and 60%.[121] There is controversy concerning the prognosis of those patients who survive the immediate post-hemorrhagic period. According to Benson,[3] there is no difference in prognosis between survivors treated surgically and those treated medically. There are reports[106] of good survival rates among patients treated by hypotensive therapy and bed-rest alone. McKissock, Paine and Walsh[74] carried out controlled studies to compare conservative with surgical measures and came to the conclusion "that there is no proof that surgical treatment of ruptured intracranial aneurysms has effectively lowered the mortality unless a large hematoma is present and can be evacuated". Walker[119] discussed the controversy that exists in the literature regarding the treatment of these lesions. Consensus of current neurosurgical opinion based on many reports[8, 22, 38, 39, 45, 63, 78, 79, 84, 88, 92, 113, 120] is that good-risk patients have a better life expectancy and lower mortality rate if they are treated surgically than if they are treated with bed-rest alone. The object of surgery is to prevent recurrence of hemorrhage. Little can be done to reverse the damage caused by rupture of the aneurysm.

GRADING OF PATIENTS WITH RUPTURED BERRY ANEURYSM

In order to evaluate the results of treatment of intracranial aneurysms it is necessary to have a system of grading the patients. Botterell's classification[63] which is most widely used is as follows:

Grade I.—Conscious with or without subarachnoid hemorrhage.

Grade II.—Drowsy without significant neurological deficit.

Grade III.—Drowsy. Semicomatose with neurological deficit.

Grade IV.—Major neurological deficit and deterioration (clot). Older patients with pre-existing degenerative and cerebrovascular disease.

Grade V.—Moribund or nearly moribund. Extensor rigidity.

Patients are given one additional grade in this classification for both hypertension and old age.

SURGICAL TECHNIQUE

A classification of surgical technique is given in Table I. A discussion of these techniques follows.

A. Extracranial Approach

1. *Carotid ligation.*—The role of carotid ligation in surgery of intracranial aneurysms is limited and many surgeons do not employ this method at all.[78, 79] Wright and Sweet[125] and Luessenhop, Mora and Sweet[64] recently reviewed this subject extensively. The objectives of this procedure are (i) to protect the aneurysm by causing a drop in the intra-arterial pressure distal to the point of ligation; (ii) to protect the aneurysm from direct pulsatile flow of blood; (iii) to lessen the tendency of circu-

*Suite 540, 2184 West Broadway, Vancouver 9, B.C.

TABLE I.—Classification of Surgical Technique

A. *Extracranial approach (procedures carried out on neck vessels)*
   1. Carotid ligation
   2. Partial carotid occlusion
   3. Vertebral artery ligation
B. *Intracranial approach (direct attack)*
   1. Occlusion of stalk of aneurysm with clip or ligature
   2. Partial clipping of aneurysmal stalk or neck
   3. Reinforcement of aneurysmal sac with muscle, cotton gauze or plastic materials
   4. Obliteration of aneurysmal sac by pilojection or coagulation with implanted electrode
   5. Flattening of whole aneurysm with a large clip
   6. Excision of aneurysmal sac
   7. Clipping of parent vessel proximal to the aneurysm, or both proximal and distal
C. *Combined intracranial and extracranial approach,* e.g. trapping of internal carotid aneurysm by ligation of internal carotid artery intracranially and carotid ligation in neck.

lating blood to deposit destructive lipids in wall of the aneurysmal sac, and (iv) to avoid intracranial procedures.

Study of stress-strain curves of aneurysm walls shows a non-linear pattern. There is a rapid increase of volume as an unstable equilibrium is reached. Carotid ligation, by diminishing blood flow, pulse pressure and blood pressure, leads to diminished expansion of the aneurysm and diminished tendency to rupture.

Carotid ligation is of some value in the following situations:

(i) Intracranial internal carotid aneurysms originating at the posterior communicating artery. McKissock, Richardson and Walsh[73] and Walsh *et al.*[120] found the results of carotid ligation in this group comparable to direct intracranial attack.

(ii) Aneurysms of petrous and cavernous portions of internal carotid artery.

(iii) As an adjunct to intracranial internal carotid ligation.

Preoperatively the patient is assessed both clinically and radiologically to establish the existence of adequate cross-circulation following carotid compression. The common carotid artery is dissected in the neck and occluded by a variety of methods:

(i) Silk ligature: This is likely to damage the intima and to result in aneurysm formation at the site of ligation,[34] or to produce a thrombus which may propagate from the damaged intima.

(ii) Tantalum band obviates the disadvantages of silk ligature, but it is not very secure and may reopen or dislodge.

(iii) Gradually occluding clamps may enable collateral circulation to develop. The clamp can be reopened if the patient shows signs of cerebral ischemia. All of the clamps protrude from the operative wound during the process of occlusion, which may take several days and infection of the wound may occur during this period. Selverstone and Crutchfield have devised clamps with detachable handles that can be removed through the wound and the clamp is left on the artery.

The major complications of carotid ligation are cerebral ischemia and carotid thrombosis. To obviate these complications, Smith[108] devised the procedure of differential carotid ligation, that is, gradual occlusion of the common carotid artery after ligation of the external carotid artery above the origin of the superior thyroid artery in order to promote enough collateral circulation to prevent thrombosis.

2. *Partial carotid occlusion.*—Ecker and Riemenschneider[24] reported a case of deliberate thrombosis of an intracranial internal carotid aneurysm by partial occlusion of the carotid artery in the neck, under radiographic control. Recently this procedure has been suggested on a hemodynamic basis[50] with the following objectives: (i) To change the pulsatile blood flow in the carotid artery to a non-pulsatile state and thus minimize the hemodynamic disturbances in the aneurysm sac. This may also disrupt resonance phenomenon that may cause the aneurysm to blow out.[49] (ii) To maintain blood flow through the carotid artery. This procedure should be considered for patients who are unable to tolerate complete carotid occlusion.

3. *Vertebral artery ligation.*—Vertebral artery ligation in the neck has no role in the treatment of intracranial aneurysms.

B. Intracranial Approach (Direct Attack)

1. *Occlusion of the stalk of the aneurysm.*—This was first performed by Dandy[18] and is still the most common form of direct attack on such aneurysms. Various kinds

of clips, such as those of Olivecrona and Mayfield, are available. These clips, made of stainless steel, silver or tantalum, are easy to apply but may slip or reopen and occasionally may tear the aneurysm stalk or the parent vessel particularly when the latter is atheromatous. Ligation of the aneurysmal stalk is preferred by some surgeons,[78] and the materials used are silk, cotton or linen thread. A ligature is more secure than a clip but is difficult to apply and if applied improperly may impair the circulation through the parent vessel. Occasionally an aneurysm may continue to fill through an aberrant vessel and it is worthwhile to open the sac after ligation of the stalk. If necessary the sac can be obliterated by muscle packing or other techniques.

2. *Partial clipping or ligation of an aneurysm.*[58]—This is indicated when the aneurysm stalk is not accessible, when the stalk is unusually broad, and when the fundus of the aneurysm presents first during dissection and ruptures. Under these circumstances a clip or a ligature may be placed across the sac. The effectiveness of this procedure is explained by the pathological studies of Crawford,[13] who found that 64% of the aneurysms rupture at the fundus, and that rupture of the stalk is rare. Exclusion of the fundus of the aneurysm alone from the circulation would provide some protection against rupture. Nyström,[81] on the basis of electron microscopical studies of aneurysms, believes that partial ligation of the aneurysm may prevent the deposition of macrophages and destructive lipids, most active in the region of the fundus. Partial clipping of the sac would create a greater disparity between the size of the sac and its orifice and, according to the observations of Black and German, would tend to promote thrombosis in the sac.[11]

3. *Reinforcement of aneurysmal sac.*—The various techniques used for this purpose are:

(i) Muscle wrapping. This method was first employed by Dott[19] and the object is to envelop the aneurysmal sac with perivascular fibrosis arising from crushed muscle. This process may take two to three weeks and recurrence of aneurysmal rupture has been reported during this period.

(ii) Gillingham[33] has used fine-mesh cotton gauze to wrap the aneurysm.

(iii) Jaeger[48] used a thin layer of cotton to wrap the aneurysm.

(iv) Selverstone[102] has had the most extensive experience in the use of synthetic materials to reinforce the aneurysm wall. After experimenting with a number of chemicals, he evolved a method in which a coat of polyvinyl-polyvinylidine latex is sprayed from an airbrush producing a thin dry flexible film intimately adherent to the adventitia of the aneurysm and the adjacent vessels. A second coat of epoxy-polyamide firmly bonds to the first coat thus providing strength while maintaining resilience and dimensional stability. These substances appear to be non-toxic and do not give off heat as they polymerize. There is, however, some reaction in the surrounding tissues leading to fibrosis and adhesion of the meninges. This may reinforce the aneurysm and thus serve a useful purpose. These materials may have an epileptogenic effect on the cerebral cortex but no long-term follow-up studies are available in human cases to settle this point.

4. *Obliteration of the aneurysmal sac.*—This can be achieved by any of the following methods:

(i) Muscle plugs introduced through an incision or tear in the aneurysm wall.

(ii) Pilojection,[30] a technique in which the shaft of a stiff mammalian hair is injected into the aneurysmal sac to produce thrombosis. Factors supposed to favour intra-aneurysmal clotting by this procedure are mechanical, associated with the presence of scales on the shaft of the hair and the negative ionic charges on the surface of the hair, which results in union with the positive calcium ions in the blood. The advantage of this technique is that it does not require complete exposure of the aneurysmal sac. A theoretical objection to it is the propagation of the blood clot from the sac of the aneurysm to the parent vessel.

(iii) Thrombosis of the aneurysmal sac induced by insertion of fine wires into the sac has been reported.[122] Coagulation of the aneurysm by insertion of fine bipolar electrodes into the sac has also been successfully performed.[35]

5. *Flattening of whole aneurysm.*—This can be achieved by pressing the aneurysm between the blades of a clip devised by Tovi.[115]

6. *Excision of aneurysmal sac.*—This is indicated when aneurysms are producing symptoms by local pressure on important structures, and is also carried out in the case of large aneurysms.[32]

7. *Clipping of parent artery.*—Occlusion of the parent artery proximal to the aneurysm is produced by the technique employed by Logue[62] for certain aneurysms of the anterior communicating artery in which the pericallosal artery on the side of the aneurysm fills from the contralateral anterior cerebral artery. The objective is to protect the aneurysm from the direct pulsatile flow of blood from the ipsilateral anterior cerebral artery. There is a danger, however, of damage to the recurrent artery of Heubner which arises from the proximal portion of the anterior cerebral artery.

Aneurysms on small arteries, such as the posterior inferior cerebellar artery, the occlusion of which will not produce disabling neurological deficit, can be excluded from circulation by clips on the parent vessel both proximal and distal to the defect.[91]

C. Combined Intracranial and Extracranial Approach

Approach to intracranial aneurysms according to location.—The location of the aneurysm is an important factor in deciding on operation and in selecting the surgical method. There is hardly any portion of the cerebrovascular tree that cannot be reached surgically. For convenience of description these aneurysms are divided into two groups: those on the internal carotid artery and its branches, and those on the vertebral-basilar system and its branches.

1. *Aneurysms from Internal Carotid Artery and its Branches*

Aneurysms arising from internal carotid arteries and their branches constitute 85% of all intracranial aneurysms.[121] With respect to treatment, the internal carotid artery aneurysms may be divided into three groups:

(i) *Supraclinoid.*—McKissock, Richardson and Walsh[73] call all of the aneurysms arising from this part of the internal carotid "posterior communicating aneurysms". In this group they include aneurysms arising from the internal carotid at the origin of the ophthalmic and anterior choroidal arteries. These authors believe that carotid ligation is as effective a treatment for these lesions as direct intracranial approach. Lougheed, Botterell and Morley[63] have reported a mortality rate of 22% for this group of aneurysms with direct surgical attack as compared with an overall mortality of 28.1% (including patients not operated upon). Posterior communicating aneurysms are best approached by a frontotemporal craniotomy and elevation of the temporal lobe.[87] Problems encountered in the management of aneurysms located at carotid bifurcation have been discussed by Björkesten.[5] Some of these aneurysms cannot be obliterated safely and are best dealt with by methods which reinforce the aneurysmal sac.

(ii) *Aneurysms in the cavernous portion of the internal carotid artery.*[54]—These are rare. Rupture may lead to carotid cavernous fistula. The usual treatment is by carotid ligation in the neck supplemented by intracranial ligation if necessary. With the development of direct approaches to the cavernous sinus[85] it may be possible to deal with these lesions by direct attack. Gallagher and Baiz[31] have reported successful treatment of these lesions by pilojection.

(iii) *Aneurysms arising in carotid canal from the petrous segment of the internal carotid artery.*—These too are rare. One case, reported by Harrison, Odom and Kunkle,[40] was treated by carotid ligation in the neck; this led to reduction in size of the sac as demonstrated by postoperative angiography and by relief of symp-

toms of involvement of the Gasserian ganglion.

Saccular aneurysms on the ophthalmic artery are quite rare. Only one such report in which the condition was definitely diagnosed and the patient operated upon has appeared in the literature.[83]

An aneurysm arising from the distal part of the anterior choroidal artery and presenting as an intraventricular mass has been reported.[111]

Anterior Cerebral Artery Aneurysms

The management of aneurysms from this artery can be divided according to location into three parts:

(i) *Aneurysms of the proximal anterior cerebral artery*[59] (portion of the artery extending from the internal carotid bifurcation to the anterior communicating artery) are rare. To get at these lesions, the internal carotid bifurcation is exposed by craniotomy and the anterior cerebral artery is followed to reach the aneurysm.

(ii) *Anterior communicating artery aneurysms* are a most difficult problem. Not only is the mortality from rupture of these aneurysms high, but it is also difficult to obliterate them by direct surgical attack. Richardson, Lane and Payne[97] have devised a method to select cases for conservative or surgical therapy. Good-risk patients treated by present surgical techniques still have a far better prognosis than those treated conservatively.[88] Some of the commonly used approaches to expose these lesions are: (a) Unilateral frontal craniotomy and retraction of the undersurface of the frontal lobe mechanically, and the use of agents to diminish cerebral volume. Poppen[90] advises resection of the medial portion of frontal lobe to expose the lesion if necessary. An alternative approach that is recommended is retraction of the frontal lobe laterally to expose the anterior communicating artery. (b) French, Zarling and Schultz[29] resect a wedge-shaped portion of the anteromedial frontal lobe, the apex being at the anterior communicating artery. This is combined with hypotensive technique and is associated with no detectable neurological deficit. This portion of the brain may be damaged in any case by the aneurysmal rupture and surgical retraction and its excision may be a safeguard against postoperative cerebral edema. (c) The most satisfactory and widely employed technique is that developed by Pool,[87] using bifrontal craniotomy under hypothermia, and staged temporary clipping starting from the internal carotid artery and converging on the aneurysm. Other surgeons[68, 109] using the same technique have reported good results. The particular advantages of this approach are wide exposure and safety. The drawbacks are loss of the sense of smell from bilateral olfactory tract disruption and complications arising from the opening of frontal sinuses.

(iii) *Pericallosal artery aneurysms*[60] are rare. They are mostly located at the genu of the corpus callosum at the bifurcation of pericallosal and callosomarginal arteries. Surgical approach is by unilateral frontal craniotomy and along the interhemispheric fissure to the superior surface of the corpus callosum. The aneurysm may be buried in the cerebral cortex or be adherent to the falx. Although there are frequent communications between the two pericallosal arteries, ligation of the main stalk of either artery is not considered to be a safe procedure.

Middle Cerebral Artery Aneurysms[72]

These are a different problem. The mortality rate for direct attack is 28.8%, and overall mortality including patients not operated upon is 38%. Middle cerebral artery aneurysms in women are different[14] in that the women are more prone to cerebral infarction after rupture and after operation, and to the development of Sylvian subarachnoid hematomas. They also have smaller and more sessile aneurysms which present greater difficulty at operation. Finally, they have a higher mortality with operation.

These aneurysms are usually exposed by the temporal approach,[90] and removal of a portion of temporal lobe is carried out to expose the aneurysm. The advantages of this technique are ease of evacuation of any intratemporal clot associated with ruptured aneurysm, and less damage to the perforating branches of the middle

cerebral artery. The disadvantage is that the aneurysm may present first and rupture before the stalk of the parent vessel can be exposed.

An alternative approach used by some surgeons is to expose the internal carotid artery at its bifurcation first and follow the middle cerebral artery up to the aneurysm. For more laterally situated aneurysms, the lateral portion of the Sylvian fissure can be opened up. This approach has the advantage of exposing the parent vessel for temporary clipping, if required, to control bleeding from the aneurysm. A disadvantage is the possibility of damage to the perforating branches of the middle cerebral artery. If the aneurysm has a well-defined stalk it can be ligated or clipped. Aneurysms located at middle-cerebral bifurcation and involving the origin of one or more important branches are best dealt with by Selverstone's technique of reinforcing the aneurysm wall.[102] The decision regarding method should be influenced by the proximity of the aneurysm to the major perforating branches (Charcot's artery), which can be demonstrated by angiography. Manipulation of the middle cerebral artery at the point of origin of a major perforating branch should be avoided.[51] It is advisable to treat these aneurysms by the techniques of reinforcing the aneurysm wall.

2. *Aneurysms from the Vertebral-Basilar System and Its Branches*

These constitute 15% of all intracranial aneurysms.[121] Most of them are located in the posterior fossa.

(i) *Vertebral artery aneurysms* are amenable to direct attack and successful attempts have been reported.[44, 80, 101]

(ii) *Basilar artery aneurysms* are best exposed by the anterior temporal approach developed by Drake.[21] It is possible to occlude the stalk of the aneurysm located at the basilar bifurcation or lower down at the point of origin of one of its branches. For an aneurysm at the basilar bifurcation with no definite stalk and with both distal posterior cerebral arteries being irrigated from the carotid system, Mount and Taveras[76] ligated the trunk of the basilar artery between the posterior cerebral and superior cerebellar arteries. Further clipping of the proximal posterior cerebral artery on each side effectively trapped the aneurysm without impairing the circulation to the territory of the posterior cerebral arteries.

(iii) *Aneurysms located on the branches of the vertebral-basilar system.*[90, 91]—Posterior cerebral artery aneurysms can be approached at the tentorial hiatus by posterior temporal approach. Aneurysms on the superior cerebellar and posterior inferior cerebellar arteries can be approached by suboccipital craniectomy on the side of the aneurysm. A portion of the cerebellum may have to be resected to uncover the aneurysm. Aneurysms on the anterior inferior cerebellar and internal auditory arteries can be approached by the same technique as for cerebellopontine-angle tumours.

Multiple Aneurysms

Poppen and Fager[93] have stressed the importance of complete bilateral angiograms where a single aneurysm has been demonstrated. These authors advocate any surgical procedure that is possible for the second aneurysm because they consider this a lesser risk than the dire consequences of recurrent hemorrhage. This problem has been reviewed recently by McKissock *et al.*[71] The reported incidence of multiple aneurysms is 13.7% in angiographic studies, and they constitute 28% of the aneurysms found at autopsy. Multiple aneurysms are associated with a higher natural mortality than single aneurysms, and recurrent hemorrhages almost always occur from the original lesion. The recurrent hemorrhages occur earlier and more frequently than in the case of single aneurysms. Only 85 out of 251 patients in McKissock's series were subjected to operation. The criteria used by these authors for selecting such cases were that the responsible lesion could be demonstrated and treated surgically where possible or that the multiple lesions identified could be treated by one operation. It is sometimes difficult to determine which of the many aneurysms has ruptured but, ac-

cording to the angiographic criteria described by Wood,[124] it should be possible to identify the ruptured aneurysm among multiple lesions in more than 95% of the cases.

Management of Unruptured Aneurysms

Sometimes aneurysms are incidental findings at angiography carried out for other reasons and show no evidence of having ruptured. At present there is no definite method to predict if a particular aneurysm will rupture in the future. According to the theory of aneurysmal rupture by resonance phenomenon proposed by Jain,[49] the size of the aneurysm is a critical factor in rupture. However, it is not yet possible to determine whether an aneurysm of a given size will rupture or not. Not enough is known about the life history of aneurysms, but there is evidence that aneurysms increase in size in the course of time.[6] This favours the treatment of these lesions whenever they are detected, because we do not know at what stage in their life they may rupture.

Large Aneurysms as Space-Occupying Lesions

There is no strict demarcation between a small and a large aneurysm. Heiskanen and Nikki[43] consider a large aneurysm as one that exceeds 1.5 cm. in posteroanterior or lateral views of the angiogram. Large aneurysms have been reported in all locations on the circle of Willis and present as intracranial space-occupying lesions. They may mimic a pituitary tumour,[123] posterior fossa tumour[52] or an intraventricular tumour.[111]

Rupture of very large aneurysms is rare and they are usually found filled with solid thrombus. They may nevertheless require treatment because of their space-occupying effect. In large aneurysms that are patent, reduction in size can be achieved by temporary occlusion of the parent vessel before ligation of the aneurysm. As an alternative, blood may be aspirated from the aneurysm with a syringe and needle after the stalk is ligated. If the collapsed aneurysm has a considerable mass it may be advisable to excise the sac.[32]

Large aneurysms filled with solid clot and producing symptoms by local pressure or by elevating intracranial pressure should be removed. This may not be possible because sometimes the aneurysm involves major vessels and cannot be excised without impairing the cerebral circulation.

Aneurysms in Pregnancy

The frequency of subarachnoid hemorrhage in pregnant women is no greater than in non-pregnant women of the same age group.[121] Pregnancy and labour appear to be of only minor significance in the etiology of rupture of intracranial aneurysms. Heiskanen and Nikki[43] conclude that the neurosurgical treatment of ruptured aneurysm in pregnancy is indicated immediately after the diagnosis has been made. The aneurysm is not an indication for the termination of the pregnancy. If the aneurysm has been treated, a normal vaginal delivery can take place. If, however, the aneurysm is not treated, cesarean section must be considered, although it is not obligatory.

Cannell[10] expressed similar views. According to him, the obstetrical management of such patients should be based on sound principles—cesarean section being employed where indicated and not routinely. As an adjunct to operation, he favoured the use of hypothermia rather than hypotension.

Aneurysms Associated with Other Lesions

Aneurysms have been reported in association with arteriovenous malformations and neoplasms.[96] A patient is more likely to die from a ruptured aneurysm than from an arteriovenous malformation and if these lesions coexist, the aneurysm should receive priority in treatment. An intact aneurysm as an incidental finding in a patient with malignant primary or secondary neoplasm of the brain may be left alone if the patient has a short life-expectancy.

TABLE II.—Complications of Ruptured Berry Aneurysms

A. *Complications due to acute rupture*
   1. Subarachnoid hemorrhage
   2. Intracerebral hemorrhage
   3. Intraventricular hemorrhage
   4. Hypothalamic lesions
   5. Epistaxis
   6. Carotid cavernous fistula
   7. Subdural hemorrhage
   8. Spasm of cerebral vessels
   9. Cerebral infarction
B. *Operative complications*
   1. Massive hemorrhage from aneurysm or parent vessel
   2. Infarction from occlusion of major vessels
   3. Brain damage from retraction
C. *Late complications*
   1. Hydrocephalus
   2. Recurrent hemorrhage from an inadequately treated aneurysm
   3. Thrombosis of aneurysm with embolization or thrombosis of parent vessel

Complications of Ruptured Berry Aneurysms

The complications of ruptured berry aneurysms are shown in Table II. A rational approach to the management of these complications requires an understanding of the pathology of various complications.

A. Complications Due to Acute Rupture

1. *Subarachnoid hemorrhage.*[107]—This is the commonest sequel of a ruptured aneurysm. It may result in elevated intracranial pressure for hours or days, which may be due either to failure of absorption of cerebrospinal fluid or to cerebral edema. Spasm of cerebral vessels may lead to cerebral ischemia which is another factor in aggravating cerebral edema. This may be responsible for the lowered state of consciousness, neuronal "fallout" and dementia, which may occur in those who survive. This state of affairs is offered as an argument against immediate surgical intervention,[78] which may add insult to injury. Some procedures, such as carotid ligation, would be inadvisable under these circumstances. A subarachnoid hematoma may form, and the common sites are between the frontal lobes, between the lips of the Sylvian fissure and in the depths of sulci.[9] These hematomas require surgical intervention. Cerebrospinal fluid drainage by lumbar puncture has no role in diminishing morbidity and may be harmful.

2. *Intracerebral hemorrhage.*—Some of the conclusions from Crompton's study[16] that are of importance in the surgical management of intracerebral hemorrhage are: (i) Intracerebral hemorrhage is more often a cause of coma than subarachnoid hemorrhage alone. (ii) There is a higher incidence of tentorial herniation leading to secondary brain-stem hemorrhage from intracerebral hemorrhage than from cerebral infarction. (iii) Previous subarachnoid hemorrhage from an aneurysm makes it more likely to rupture into the brain at a subsequent episode of bleeding. (iv) Anterior communicating and anterior cerebral aneurysms result in hematoma formation more often than aneurysms at other sites and commonly produce hematoma in the frontal lobe. Middle cerebral artery aneurysms more frequently produce hematomas in the external capsule and less frequently in the temporal lobe. Posterior communicating aneurysms rupture into the temporal lobe, whereas aneurysms at the bifurcation of internal carotid artery often rupture into frontal lobes.

The presence of intracerebral clot is considered to be one of the indications for early operative intervention, because some of the neurological deficit (e.g. hemiplegia) may be due to pressure rather than to destruction of nervous tissue and evacuation of the clot may lead to some degree of recovery. The evacuation of the clot may also facilitate the exposure of the aneurysm. A decision as to whether the aneurysm is clipped at the same time as the evacuation of the clot depends on the general condition of the patient.

3. *Intraventricular hemorrhage.*—This is often associated with fatal aneurysmal rupture. The fate of blood in the ventricular system has been described by Sanders.[100] There is no evidence that the presence of blood in the ventricles alone would cause more morbidity than subarachnoid hemorrhage alone. More important in determining the morbidity is the route by which blood reaches the ventricles. Aneurysms of the anterior portion of the circle of Willis may damage important hypothalamic structures along their track of rupture into the ventricles. There is no definite evidence in

the literature that the evacuation of intraventricular clot improves the mortality or morbidity of patients with ruptured aneurysms.

4. *Hypothalamic lesions.*—These lesions were a common finding in cases of ruptured aneurysms examined at autopsy by Crompton.[15] They are commonly associated with aneurysms of the anterior cerebral artery. Further complications of hypothalamic lesions such as gastric hemorrhage have been reported. Clinical evidence of severe hypothalamic damage at the time the patient presents with ruptured aneurysm would be a point against immediate operative intervention.

5. *Epistaxis.*—This is a rare but known complication of internal carotid aneurysms eroding into the sphenoid sinus. A case has been reported[117] in which successful treatment was carried out by trapping the aneurysm by carotid ligation in the neck as well as intracranially.

6. *Carotid cavernous fistula.*—As a complication of rupture of an internal carotid aneurysm into the cavernous sinus, carotid cavernous fistula is a problem in management.[23] Some of the procedures for this complication are (i) trapping by carotid ligation in the neck, *plus* intracranial internal carotid ligation *plus* ophthalmic artery ligation; (ii) occlusion of the cavernous segment of the internal carotid artery by a variety of procedures such as packing with muscle; (iii) a direct approach to these lesions by opening the cavernous sinus, as made by Parkinson[85] recently.

7. *Subdural hematoma.*[110]—This is a rare complication of ruptured intracranial aneurysms. Small amounts of blood may resolve spontaneously but larger amounts would require surgical intervention.

8. *Spasm of cerebral vessels.*—This problem is receiving increasing attention.[27, 61, 89] Spasm is rarely seen in subarachnoid hemorrhage other than that due to ruptured aneurysm. It may be due to vasospastic substances such as serotonin released from the blood clot,[107] but serotonin antagonists have not been proved to relieve it. It is usually confined to the segment of the artery contiguous to the aneurysm. Mechanical and irritative factors caused by the distension of the aneurysm sac may be responsible. Theoretically, obliteration of the aneurysm would remove the source of such noxious stimuli but attempts to do so by direct surgical approach may involve manipulating affected vessels and aggravating spasm. Allcock and Drake[1] feel that spasm is the main cause of postoperative morbidity and mortality in otherwise uncomplicated cases of ruptured aneurysms. Local application of phentolamine (Rogitine) and papaverine has been reported to be effective in relieving spasm.[61] The relation of spasm to hypothermia is also being investigated.

9. *Cerebral infarction.*[4]—This is a major cause of mortality and is found in the territory of distribution of the parent artery of the aneurysm as well as in other areas not supplied by the aneurysm-bearing artery. There is no indication at present that an operation can help a moribund patient with extensive cerebral infarction. Only conservative measures and those directed at diminishing cerebral edema are indicated.

B. *Operative Complications*

1. *Massive hemorrhage.*—Massive hemorrhage from the "blowing off" of the aneurysm may occur at operation. Hypotensive anesthesia lessens the bleeding. The proximal vessel if available can be occluded temporarily. This problem can be handled by getting the fundus of the aneurysm into the suction tip and placing a clip across the stalk.[90] Hemorrhage from the parent vessel may occur when the aneurysm tears off from it. If this is anticipated and the patient is operated upon under hypothermia, the vessel can be repaired by the technique described by Carton, Heifetz and Kessler.[11] Under emergency circumstances, Simmons, Portnoy and Gurdjian[105] have controlled hemorrhage from the internal carotid artery by a pledget of muscle wedged between the artery and the sphenoid bone.

2. *Infarction from occlusion of major vessels.*—Infarction from the temporary or permanent occlusion of major vessels can be minimized by good preoperative assessment of collateral circulation, and by operating under hypothermia. Another cause of infarction may be embolization from the aneurysmal sac during manipulation at operation.[89]

3. *Brain damage from retraction.*—This damage can be minimized by careful assistants, use of agents to decrease cerebral volume and finally by resecting rather than retracting certain parts of the brain.

C. *Late Complications*

1. *Hydrocephalus.*—Extraventricular obstructive hydrocephalus may result from subarachnoid hemorrhage and would require a cerebrospinal fluid shunting procedure.[104] Ventricular dilatation is supposed to be secondary to adhesive arachnoidal reaction, leading to obstruction of cerebrospinal fluid pathways. Since the fibrosis of meninges takes at least 10 to 14 days to develop, the appearance of hydrocephalus is delayed and more commonly follows repeated subarachnoid hemorrhages.

2. *Recurrent hemorrhage from an inadequately treated aneurysm.*—This is more common following indirect procedures such as carotid ligation. Follow-up angiography may demonstrate that the clip placed across the stalk has opened up or slipped leading to refilling of the aneurysm sac.[1] Recurrent hemorrhages may be from a traumatic aneurysm which has developed as a sequel to an operation for occlusion of the original aneurysm.[25] Radioactive isotopes have proved useful in detecting fresh subarachnoid hemorrage.[98]

3. *Thrombosis of aneurysm with embolization or thrombosis of parent vessel.*—Retrograde thrombosis from an aneurysm spreading to the internal carotid artery has been reported.[26] It is difficult to remove such a thrombus. If there is evidence that embolization has occurred from the thrombus in the aneurysmal sac, the stalk should be ligated or the parent vessel distal to the aneurysm should be occluded.

TIMING OF SURGERY

The timing of surgery is a controversial subject. Most surgeons[17, 63, 74] favour early angiography and operative intervention in those patients who are known to harbour a clot and who are not moribund. Other factors, such as the medical condition of the patient (i.e. fitness to undergo an operation) and the availability of technical facilities, are important in making a decision. The main object of early intervention is to prevent recurrence of subarachnoid hemorrhage, the peak incidence of which is in the second week following the first hemorrhage.[121]

The Swedish school[78, 79] does not believe in operating during the acute bleeding state, because there is higher operative mortality in patients operated upon within the first three weeks of aneurysmal rupture. Operative mortality during this period is not less than the mortality of all persons with ruptured berry aneurysms. They consider the degree of consciousness of the patient to be more important than the time factor. As soon as the patient more or less recovers from the effect of aneurysmal rupture, an operation can be performed with low mortality and morbidity. Patients who show no evidence of recovery are saved the trouble of unnecessary surgery. Criticism of this approach is that some of the patients will have a fatal recurrence of hemorrhage before an operation is performed.

Hunt, Meagher and Barnes[45] feel that Grade I and Grade II (Botterell's classification) patients should have an angiogram and an operation as soon as possible. In Grade III and Grade IV they prefer to delay the operation unless hemorrhage recurs in spite of the conservative measures. Lougheed *et al.*[63] also believe in observing patients in Grade III and Grade IV categories for one week and then operating if there are signs of recovery.

ROLE OF RADIOLOGY

The role of radiology in the study of intracranial aneurysms has been reviewed by Bull.[9] Although it is possible to localize an aneurysm in certain cases on clinical grounds,[118] radiological assessment is essen-

tial. Plain radiographs of the skull may be helpful if there is local bony erosion or if the aneurysm is calcified. Angiography is a necessary preoperative procedure for the following reasons: (i) to show the exact location of the aneurysm; (ii) to determine the shape and size of the aneurysmal sac; (iii) to detect multiple aneurysms; (iv) to rule out other causes of subarachnoid hemorrhage or other lesions associated with an aneurysm; (v) to determine if the aneurysm is intact or ruptured; (vi) to assess the complications of aneurysmal rupture—intracerebral hemorrhage, subdural hematoma and spasm of cerebral vessels, and (vii) to demonstrate anatomical variations of the cerebral vessels associated with aneurysms.

The usual routine is to perform bilateral carotid angiography and, if this is negative, to carry out vertebral angiography after an interval of 24 hours. This routine may be modified under the following circumstances: (i) if the clinical localization of the aneurysm is to the vertebral basilar system, vertebral angiography may be performed first; (ii) if the patient has definite subarachnoid hemorrhage and the aneurysm demonstrated on carotid angiography does not appear to be responsible for it, vertebral angiography should be carried out.

The timing of angiography is related to the timing of surgery. Those who believe in early operation perform an immediate angiography on admission. Those who believe in delayed operation postpone this procedure until the patient begins to recover from the effects of the first episode of bleeding. Postoperative angiography also reveals important information as pointed out by Allcock and Drake.[1]

Limitations of Angiography

1. Accuracy in diagnosing aneurysms by this method has been reported to be 89% as verified by autopsy and it is predicted that it will reach a potential accuracy of 96% with refinements in angiographic techniques.[86] Spasm and thrombosis may prevent filling of an aneurysm.

2. Aneurysms less than 2 mm. in size cannot be diagnosed with certainty on angiography, nor is it possible to show the minute aneurysms described by Hassler,[41] which occurred in 17% of brains selected at random at autopsy.

3. The differential diagnosis of the following complications of ruptured aneurysm (intracerebral hemorrhage, cerebral infarction with edema, and subarachnoid hematoma) is not usually possible.

4. Carotid circulation may not be demonstrable owing to elevated intracranial pressure[95] associated with aneurysmal rupture and subarachnoid hemorrhage.

5. Rupture of aneurysms at the time of angiography has been reported[47] but is considered to be coincidental. There is no proof that angiography causes enough hemodynamic disturbances to rupture the aneurysm.

6. Complications of angiography are rare and only of a minor consideration.

7. False diagnosis, e.g. funnel-shaped dilatation at the origin of the posterior communicating artery may be mistaken for a true aneurysm if the posterior communicating artery does not fill.[28]

Anesthesia and Adjunctive Measures for Operation on Intracranial Aneurysms

This subject has been reviewed by Marshall[67] whose classification of various techniques is shown in Table III.

TABLE III.—Classification of Adjunctive Measures for Surgery of Intracranial Aneurysms

1. Induced hypotension
2. Use of hypertonic solutions to diminish cerebral volume
3. Passive hyperventilation
4. Controlled ventilation with positive and negative pressure phases
5. Induced hypothermia—medium
   —deep . . . local
   . . . total body

Induced hypotension is a valuable adjunct to direct surgical attack on aneurysms and was in use before the introduction of other methods. Some surgeons have continued to operate upon patients with intracranial aneurysms, using this technique combined with cerebrospinal fluid drainage, and find it quite satisfactory. This procedure requires cautious control.

Among the hypertonic solutions, urea[53] has been used most widely to diminish cerebral volume and facilitate exposure of the aneurysms. There is danger however of a rebound phenomenon with this agent and more recently mannitol[103] has been introduced for the same purpose. It is not considered advisable to combine hypotension with this technique.

Hypothermia is the most widely used and important adjunct to the surgery of intracranial aneurysms.[8] Its role has, as yet, not been settled. It is considered to be a safe procedure and, according to Boba,[7] does not result in any added morbidity or mortality. Karlsberg and Adams[55] make a point in favour of hypothermia; in most of their cases, it caused the aneurysm to collapse to one-half of the preoperative size and helped to control the bleeding from the ruptured aneurysm. They felt that complications associated with this procedure are not serious enough to lead to the discard of it. McKissock *et al.*[70] obtained a reduction in mortality with use of hypothermia in patients operated upon within three days after rupture of aneurysm. Maspes and Marini[69] are of the opinion that the use of hypothermia and interruption of cerebral blood flow facilitates direct radical treatment of intracranial aneurysms. Hamby,[37] on the basis of comparison of patients operated upon under hypothermia with a series without hypothermia by the same group of surgeons, found that hypothermia did not improve the survival rate. Moderate hypothermia will provide 10 to 12 minutes' protection against anoxia from total vascular occlusion.

For more prolonged occlusion two methods have been developed.

1. Profound hypothermia with circulatory arrest and extracorporeal circulation has been employed by Uihlein *et al.*[116] at the Mayo Clinic. MacCarty, Michenfelder and Uihlein[66] have recently reviewed three years' experience with this technique but they arrived at no conclusion regarding the efficiency of this technique because of the circumstances involved in the selection of patients and the many procedures used. Based on their experience, Drake *et al.*[20] have stated that "these techniques create enough additional hazards that they are not warranted in cases where the aneurysms can be obliterated by conventional means. The complications of postoperative arterial spasm and ischemic brain damage is not prevented by this technique".

2. Local brain cooling can be achieved by two methods: (i) Vascular.[57]—With this technique the brain is perfused with blood cooled by a heat exchanger via the carotid artery. (ii) Extravascular ventricular perfusion.[82]—With this technique the cerebral ventricles are perfused with a cold solution.

Future Outlook in Aneurysm Surgery

Surgery of the intracranial aneurysm is still in a state of evolution and new techniques are continually being developed. Beginning with carotid ligation, neurosurgeons have progressed to the stage where the best method of treating these lesions is considered to be their exclusion from the circulation. Various techniques to achieve this have been discussed. Newer and better techniques are becoming available.

There is some promise in the intra-arterial approach to the vascular malformations. Luessenhop and Velasquez[65] have shown that intraluminal manipulation of the intracranial arteries about the circle of Willis is possible technically by catheterization and artificial embolization. Further developments in this technique may make it possible to occlude the orifice of an intracranial internal carotid aneurysm by an artificial embolus introduced into the carotid artery in the neck. Another technique worthy of mention is that of angiotactic surgery,[99] deposition of a sleeve of plastic material inside the lumen of a vessel by a device inserted at a distance into the vessel. The limitation of application of this device to internal carotid aneurysms lies in the difficulty of navigating the curves of the carotid siphon. With further developments in this technique, it may be possible to deposit a sleeve of plastic material in the lumen of the internal carotid artery at the level of the aneurysm to exclude the aneurysmal orifice.

Mullan *et al.*[77] have developed techniques to induce thrombosis in the aneurysm by a probe inserted with stereotactic guidance through a burr hole. Refinements in the techniques of small-vessel anastomosis may enable the aneurysm-bearing segment of the artery to be resected and reanastomosed to maintain the circulation through the parent vessel.[12, 46]

In the development of adjuncts to surgery, considerable effort is being made to improve the techniques of local cerebral cooling so as to enable the surgeon to occlude the blood supply to the brain for longer periods safely and to allow him to handle the intracranial contents with more ease, less damage and less anxiety.

References

1. Allcock, J. M. and Drake, C. G.: Postoperative angiography in cases of ruptured intracranial aneurysm, *J. Neurosurg.*, **20:** 752, 1963.
2. Austin, G. M. and McMahon, L.: Physiologic considerations of the development and treatment of intracranial aneurysms, *Surg. Forum*, **14:** 416, 1963.
3. Benson, D. F.: Conservative vs. surgical treatment of cerebral aneurysms, *Neurology (Minneap.)*, **8:** 852, 1958.
4. Birse, S. H. and Tom, M. L.: Incidence of cerebral infarction associated with ruptured intracranial aneurysms, *Neurology (Minneap.)*, **10:** 101, 1960.
5. Björkesten, G. A. F.: Arterial aneurysms of the internal carotid artery and its bifurcation: an analysis of 69 aneurysms treated mainly by direct surgical attack, *J. Neurosurg.*, **15:** 400, 1958.
6. Björkesten, G. and Troup, P. H.: Changes in size of intracranial arterial aneurysms, *J. Neurosurg.*, **19:** 583, 1962.
7. Boba, A.: Hypothermia: appraisal of risk in 110 consecutive patients, *J. Neurosurg.*, **19:** 924, 1962.
8. Botterell, E. H. *et al.*: Hypothermia in surgical treatment of ruptured intracranial aneurysms, *J. Neurosurg.*, **15:** 4, 1958.
9. Bull, J. W. D.: Contribution of radiology to the study of intracranial aneurysms, *Brit. Med. J.*, **2:** 1701, 1962.
10. Cannell, D. E.: Subarachnoid haemorrhage in pregnancy, *Proc. Roy. Soc. Med.*, **52:** 950, 1959.
11. Carton, C. A., Heifetz, M. D. and Kessler, L. A.: Patching of intracranial internal carotid artery in man using a plastic adhesive (Eastman 910 adhesive), *J. Neurosurg.*, **19:** 887, 1962.
12. Carton, C. A. *et al.*: Experimental studies in the surgery of small blood vessels. II. Patching of arteriotomy using plastic adhesive, *J. Neurosurg.*, **18:** 188, 1961.
13. Crawford, T.: Some observations on the pathogenesis and natural history of intracranial aneurysms, *J. Neurol. Neurosurg. Psychiat.*, **22:** 259, 1959.
14. Crompton, M. R.: Pathology of ruptured middle-cerebral aneurysms with special reference to difference between sexes, *Lancet*, **2:** 421, 1962.
15. Crompton, M. R.: Hypothalamic lesions following rupture of cerebral berry aneurysms, *Brain*, **86:** 301, 1963.
16. Crompton, M. R.: Intracerebral haematoma complicating ruptured cerebral berry aneurysm, *J. Neurol. Neurosurg. Psychiat.*, **25:** 378, 1962.
17. Dandy, W. E.: Intracranial arterial aneurysms, Comstock Publishing Co., Inc., Ithaca, N.Y., 1944, p. 147.
18. Dandy, W. E.: Intracranial aneurysm of internal carotid artery cured by operation, *Ann. Surg.*, **107:** 654, 1938.
19. Dott, N. M.: Intracranial aneurysms: cerebral arterio-radiography: surgical treatment, *Edinb. Med. J.*, **40:** 219, 1933.
20. Drake, C. G. *et al.*: The use of extracorporeal circulation and profound hypothermia in the treatment of ruptured intracranial aneurysm, *J. Neurosurg.*, **21:** 575, 1964.
21. Drake, C. G.: Bleeding aneurysms of the basilar artery: direct surgical management in four cases, *J. Neurosurg.*, **18:** 230, 1961.
22. Drake, C. G. and Jory, T. A.: Spontaneous intracranial haemorrhage: a review of investigation and treatment in 189 cases, *Canad. J. Surg.*, **4:** 4, 1960.
23. Echols, D. H. and Jackson, J. D.: Carotid-cavernous fistula: a perplexing surgical problem, *J. Neurosurg.*, **16:** 619, 1959.
24. Ecker, A. and Riemenschneider, P.: Deliberate thrombosis of intracranial arterial aneurism by partial occlusion of carotid artery with arteriographic control; preliminary report of a case, *J. Neurosurg.*, **8:** 348, 1951.
25. Finkelmeyer, H.: Ein Säck förmiges aneurysma der A., cerebri media postoperative Komplikation, *Zbl. Neurochir.*, **15:** 302, 1955.
26. Fisher, R. G. and Goran, A.: Thrombosis of intracranial aneurysm and cervical portion of internal carotid artery in a child, *J. Neurosurg.*, **18:** 698, 1961.
27. Fletcher, T. M., Taveras, J. M. and Pool, J. L.: Cerebral vasospasm in angiography for intracranial aneurysms, *A.M.A. Arch. Neurol.*, **1:** 38, 1959.
28. Fox, J. L., Baiz, T. C. and Jakoby, R. R.: Differentiation of aneurism from infundibulum of the posterior communicating artery, *J. Neurosurg.*, **21:** 135, 1964.
29. French, L. A., Zarling, M. E. and Schultz, E. A.: Management of aneurysms of the anterior communicating artery, *J. Neurosurg.*, **19:** 870, 1962.
30. Gallagher, J. P.: Pilojection for intracranial aneurysms; report of progress, *J. Neurosurg.*, **21:** 129, 1964.
31. Gallagher, J. P. and Baiz, T.: Pilojection for carotid aneurysm in the cavernous sinus, *J. A. M. A.*, **188:** 1156, 1964.
32. Gibbs, J. R.: On the advantages of opening certain intracranial aneurysms, *J. Neurol. Neurosurg. Psychiat.*, **20:** 165, 1957.
33. Gillingham, F. J.: Management of ruptured intracranial aneurysm, *Ann. Roy. Coll. Surg. Eng.*, **23:** 89, 1958.

34. Gross, S. W. and Holzman, A.: Aneurysm of common carotid artery in neck following partial ligation for intracranial aneurysm, *J. Neurosurg.*, **11:** 209, 1954.
35. Guillaume, J., Mazars, G. and Pansini, A.: Essais de coagulation intra-cavitaire des anévrysmes endocraniens, *Neurochirurgie*, **3:** 226, 1957.
36. Hamby, W. B.: Intracranial aneurysms, Charles C Thomas, Springfield, Ill., 1952, p. 564.
37. Hamby, W. B.: Intracranial surgery for aneurysm-effect of hypothermia upon survival, *J. Neurosurg.*, **20:** 41, 1963.
38. Hamby, W. B.: Intracranial surgical procedures in subarachnoid hemorrhage and intracranial aneurysms, *In*: Pathogenesis and treatment of cerebrovascular disease, edited by W. S. Fields, Charles C Thomas, Springfield, Ill., 1960, p. 496.
39. Hamilton, J. G. and Falconer, M. A.: Immediate and late results of surgery in cases of saccular intracranial aneurysms, *J. Neurosurg.*, **16:** 514, 1959.
40. Harrison, T. H., Odom, G. L. and Kunkle, E. C.: Internal carotid aneurysm arising in the carotid canal, *Arch. Neurol. (Chicago)*, **8:** 328, 1963.
41. Hassler, O.: Morphological studies on large cerebral arteries, with reference to aetiology of subarachnoid haemorrhage, *Acta Psychiat. Scand.*, Suppl. **154:** 1, 1961.
42. Heiskanen, O. and Nikki, P.: Rupture of intracranial arterial aneurysm during pregnancy, *Acta Neurol. Scand.*, **39:** 202, 1963.
43. Heiskanen, O. and Nikki, P.: Large intracranial aneurysms, *Acta Neurol. Scand.*, **38:** 195, 1962.
44. Hook, O., Norlen, G. and Guzman, J.: Saccular aneurysms of vertebral-basilar arterial system; a report of 28 cases, *Acta Neurol. Scand.*, **39:** 271, 1963.
45. Hunt, W. E., Meagher, J. W. and Barnes, J. E.: The management of intracranial aneurysm, *J. Neurosurg.*, **19:** 34, 1962.
46. Jacobson, J. H., 2nd *et al.*: Microsurgery as an aid to middle cerebral artery endarterectomy, *J. Neurosurg.*, **19:** 108, 1962.
47. Jackson, I. R., Tindall, G. T. and Nashol, D. B. S., Jr.: Rupture of an intracranial aneurysm during carotid arteriography: a case report, *J. Neurosurg.*, **17:** 333, 1960.
48. Jaeger, R.: Aneurysm of intracranial carotid artery: syndrome of frontal headache with oculomotor nerve paralysis, *J. A. M. A.*, **142:** 304, 1950.
49. Jain, K. K.: Mechanism of rupture of intracranial saccular aneurysms, *Surgery*, **54:** 347, 1963.
50. Jain, K. K.: Partial carotid occlusion as treatment for intracranial internal carotid aneurysms, *Canad. Med. Ass. J.*, **88:** 247, 1963.
51. Jain, K. K.: Some observations on the anatomy of the middle cerebral artery, *Canad. J. Surg.*, **7:** 134, 1964.
52. Jane, J. A.: A large aneurysm of posterior inferior cerebellar artery in a 1-year-old child, *J. Neurosurg.*, **18:** 245, 1961.
53. Javid, M.: Urea in intracranial surgery: a new method, *J. Neurosurg.*, **18:** 51, 1961.
54. Jefferson, G.: On saccular aneurysms of the internal carotid artery in the cavernous sinus, *Brit. J. Surg.*, **26:** 267, 1938.
55. Karlsberg, P. and Adams, J. E.: Value of hypothermia and arterial occlusion in the treatment of intracranial aneurysms, *J. Neurosurg.*, **19:** 665, 1962.
56. Krayenbuhl, H. and Yasargil, M. G.: Das Hirnaneurysma, J. R. Geigy, S.A., Basel, 1958, p. 143.
57. Kristiansen, K. and Lund, I.: Experiences with selective cooling of the brain, *Acta Chir. Scand.*, Suppl. **253:** 151, 1960.
58. Laitinen, L. and Troup, P. H.: Reliability of partial ligation in treatment of intracranial arterial aneurysm, *Acta Neurol. Scand.*, **37:** 62, 1961.
59. Laitinen, L.: Aneurysms of the proximal portion of the anterior cerebral artery, *Acta Pyschiat. Scand. (Kbh.)*, **36:** 141, 1961.
60. Laitinen, L. and Snellman, A.: Aneurysms of the pericallosal artery: a study of 14 cases verified angiographically and treated mainly by direct surgical attack, *J. Neurosurg.*, **17:** 447, 1960.
61. Lende, R. A.: Local spasm in cerebral arteries, *J. Neurosurg.*, **17:** 90, 1960.
62. Logue, V.: Surgery in spontaneous subarachnoid haemorrhage; operative treatment of aneurysms on the anterior cerebral and anterior communicating artery, *Brit. Med. J.*, **1:** 473, 1956.
63. Lougheed, W. M., Botterell, E. H. and Morley, T. P.: Results of direct attack in surgical management of internal carotid and middle cerebral aneurysms, *Clin. Neurosurg.*, **9:** 193, 1963.
64. Luessenhop, A. J., Mora, F. and Sweet, W. H.: Subarachnoid hemorrhage, intracranial aneurysms and arteriovenous anomalies; treatment by cervical carotid occlusion, *In*: Pathogenesis and treatment of cerebrovascular disease, edited by W. S. Fields, Charles C Thomas, Springfield, Ill., 1960, p. 516.
65. Luessenhop, A. J. and Velasquez, A. C.: Observations on the tolerance of intracranial arteries to catheterization, *J. Neurosurg.*, **21:** 85, 1964.
66. MacCarty, C. S., Michenfelder, J. D. and Uihlein, A.: Treatment of intracranial vascular disorders with aid of profound hypothermia and total circulatory arrest: three years' experience, *J. Neurosurg.*, **21:** 372, 1964.
67. Marshall, B. M.: Anesthesia for intracranial aneurysm surgery, *Clin. Neurosurg.*, **9:** 142, 1963.
68. Maspes, P. E. and Marini, G.: Aneurysms of anterior communicating artery; results of direct surgical treatment, *Acta Neurochir. (Wien)*, **11:** 479, 1963.
69. Maspes, P. E. and Marini, G.: Intracranial aneurysms: results of direct surgical treatment with special reference to the use of moderate hypothermia and circulatory arrest, *J. Neurosurg.*, **21:** 284, 1964.
70. McKissock, W., Paine, K. W. E. and Walsh, L. S.: Value of hypothermia in the surgical treatment of ruptured intracranial aneurysms, *J. Neurosurg.*, **17:** 700, 1960.
71. McKissock, W., Richardson, A. and Walsh, L.: Multiple intracranial aneurysms, *Lancet*, **1:** 623, 1964.
72. McKissock, W., Richardson, A. and Walsh, L.: Middle-cerebral aneurysms: further re-

sults in the controlled trial of conservative and surgical treatment of ruptured intracranial aneurysms, *Lancet*, **2:** 417, 1962.

73. McKissock, W., Richardson, A. and Walsh, L.: "Posterior-communicating" aneurysms; a controlled trial of the conservative and surgical treatment of ruptured aneurysms of the internal carotid artery at or near the point of origin of the posterior communicating artery, *Lancet*, **1:** 1203, 1960.
74. McKissock, W., Paine, K. W. E. and Walsh, L. S.: An analysis of treatment of ruptured intracranial aneurysms — a report of 772 consecutive cases, *J. Neurosurg.*, **17:** 762, 1960.
75. Messer, H. D., Strenger, L. and McVeety, H. J.: Use of plastic adhesives for reinforcement of a ruptured intracranial aneurysm, *J. Neurosurg.*, **20:** 360, 1963.
76. Mount, L. A. and Taveras, J. M.: Ligation of basilar artery in treatment of aneurysm at the basilar-artery bifurcation, *J. Neurosurg.*, **19:** 167, 1962.
77. Mullan, S. *et al.*: An experimental approach to the problem of cerebral aneurysms, J. *Neurosurg.*, **21:** 838, 1964.
78. Norlen, G.: Some aspects of surgical treatment of intracranial aneurysms, *Clin. Neurosurg.*, **9:** 214, 1963.
79. Norlen, G. and Olivecrona, H.: Treatment of aneurysms of circle of Willis, *J. Neurosurg.*, **10:** 404, 1953.
80. Norlen, G. and Paly, S. N.: Aneurysms of vertebral artery: report of two operative cases, *J. Neurosurg.*, **17:** 830, 1960.
81. Nyström, S. H. M.: Development of intracranial aneurysms as revealed by electron microscopy, *J. Neurosurg.*, **20:** 329, 1963.
82. Ommaya, A. K. and Baldwin, M.: Extravascular local cooling of the brain in man, *J. Neurosurg.*, **20:** 8, 1963.
83. Parkinson, D., Jain, K. K. and Johnston, J. B.: Saccular aneurysm of the ophthalmic artery; report of an unusual case, *Canad. J. Surg.*, **4:** 229, 1961.
84. Parkinson, D.: The problem of spontaneous subarachnoid hemorrhage with proven aneurysms, *J. Neurosurg.*, **12:** 565, 1955.
85. Parkinson, D.: Collateral circulation of cavernous carotid artery: anatomy, *Canad. J. Surg.*, **7:** 251, 1964.
86. Perrett, L. V. and Bull, J. W. D.: Some aspects of subarachnoid haemorrhage: a symposium. III. The accuracy of radiology in demonstrating ruptured intracranial aneurysms, *Brit. J. Radiol.*, **32:** 85, 1959.
87. Pool, J. L.: Aneurysms of the anterior communicating artery: bifrontal craniotomy and routine use of temporary clips, *J. Neurosurg.*, **18:** 98, 1961.
88. Pool, J. L.: Timing and techniques in the intracranial surgery of ruptured aneurysms of the anterior communicating artery, *J. Neurosurg.*, **19:** 378, 1962.
89. Pool, J. L. and Fletcher, T. A.: Cerebral vasospasm—clinical and experimental evidence, *J. A. M. A.*, **167:** 1599, 1958.
90. Poppen, J. L.: Atlas of neurosurgical techniques, W. B. Saunders Company, Philadelphia, 1960.
91. Poppen, J. L.: Vascular surgery of the posterior fossa, *Clin. Neurosurg.*, **6:** 198, 1959.
92. Poppen, J. L. and Fager, C. A.: Intracranial aneurysms — results of surgical treatment, *J. Neurosurg.*, **17:** 283, 1960.
93. Poppen, J. L. and Fager, C. A.: Multiple intracranial aneurysms, *J. Neurosurg.*, **16:** 581, 1959.
94. Lang, E. F. and Poppen, J. L.: Aneurysms of anterior communicating artery; results of surgical treatment, *Surg. Clin. N. Amer.*, **42:** 793, 1962.
95. Pribram, H. F. W.: Angiographic appearances in acute intracranial hypertension, *Neurology (Minneap.)*, **11:** 10, 1961.
96. Reigh, E. E. and Lemmen, L.: Cerebral aneurysms with other intracranial pathology, *J. Neurosurg.*, **17:** 469, 1960.
97. Richardson, A. E., Lane, J. A. and Payne, P. M.: Assessment of natural history of anterior communicating aneurysms, *J. Neurosurg.*, **21:** 266, 1964.
98. Rothenberg, S. F., Penka, E. J. and Garrity, R. W.: A new isotope method for detecting continued or recurrent bleeding after subarachnoid hemorrhage, *J. Neurosurg.*, **5:** 215, 1958.
99. Rothenberg, S. F., Penka, E. J. and Conway, L. W.: Angiotic surgery: preliminary studies, *J. Neurosurg.*, **19:** 877, 1962.
100. Sanders, E.: A study of primary, immediate or direct hemorrhage into the ventricles of the brain, *Amer. J. Med. Sci.*, **82:** 85, 1881.
101. Sedzimir, C. B.: Surgical treatment of aneurysms of vertebral artery, *J. Neurosurg.*, **20:** 597, 1963.
102. Selverstone, B.: Treatment of intracranial aneurysms with adherent plastics, *Clin. Neurosurg.*, **9:** 201, 1963.
103. Shenkin, H. A., Goluboff, B. and Haft, H.: The use of mannitol for the reduction of intracranial pressure in intracranial surgery, *J. Neurosurg.*, **19:** 897, 1962.
104. Shulman, K. *et al.*: Recognition and treatment of hydrocephalus following spontaneous subarachnoid hemorrhage, *J. Neurosurg.*, **20:** 1040, 1963.
105. Simmons, D. R., Portnoy, H. D. and Gurdjian, E. S.: Packing with muscle of internal carotid artery for aneurysm with immediate occlusion and subsequent establishment of flow, *J. Neurosurg.*, **20:** 1000, 1963.
106. Slosberg, P. S.: Medical treatment of intracranial aneurysms, *Neurology (Minneap.)*, **10:** 1085, 1960.
107. Smith, B.: Cerebral pathology in subarachnoid haemorrhage, *J. Neurol. Neurosurg. Psychiat.*, **26:** 535, 1963.
108. Smith, F. P.: Differential carotid ligation for supraclinoid arterial cerebral aneurysms, *J. Neurosurg.*, **19:** 787, 1962.
109. Speakman, T. J. and Barlass, J. L.: The bifrontal approach to aneurysms of the anterior communicating artery, *Canad. J. Surg.*, **6:** 237, 1963.
110. Strang, R. R., Tovi, D. and Hugosson, R.: Subdural hematomas resulting from rupture of intracranial arterial aneurysms, *Acta Chir. Scand.*, **121:** 345, 1961.
111. Strully, K. J.: Successful removal of intraventricular aneurysm of the choroidal artery, *J. Neurosurg.*, **12:** 317, 1955.
112. Sugar, O. and Tsuchiya, G.: Plastic coating of intracranial aneurysms with "EDH-adhesive", *J. Neurosurg.*, **21:** 114, 1964.

113. Tappura, M.: Prognosis of subarachnoid haemorrhage: a study of 120 patients with unoperated intracranial arterial aneurysms and 267 patients without vascular lesions demonstrable in bilateral carotid angiograms, *Acta Med. Scand.*, **173:** (Suppl. 392) 1, 1962.
114. Todd, E. M. *et al.*: Plastic jackets for certain intracranial aneurysms, *J. A. M. A.*, **179:** 935, 1962.
115. Tovi, D.: New instrument to obliterate intracranial saccular aneurysm, *J. Neurosurg.*, **12:** 525, 1955.
116. Uihlein, A. *et al.*: Operations on intracranial aneurysms with induced hypothermia below 15° and total circulatory arrest, *J. Neurosurg.*, **19:** 237, 1962.
117. Voris, H. C. and Basile, J. X. R.: Recurrent epistaxis from aneurysm of the internal carotid artery: case report with cure by operation, *J. Neurosurg.*, **18:** 841, 1961.
118. Walker, A. E.: Clinical localization of intracranial aneurysms and vascular anomalies, *Neurology (Minneap.)*, **6:** 79, 1956.
119. Walker, A. E.: Subarachnoid hemorrhage and intracranial aneurysms medical or surgical, *Surgery*, **41:** 509, 1957.
120. Walsh, L. S. *et al.*: Experience in medical and surgical treatment of ruptured intracranial aneurysms, *Ass. Res. Nerv. Ment. Dis. Proc.*, In press.
121. Walton, J. N.: Subarachnoid haemorrhage, E. & S. Livingstone, Ltd., Edinburgh, 1956, p. 350.
122. Werner, S. C., Blakemore, A. H. and King, B. G.: Aneurysms of internal carotid artery within skull: wiring and electrothermic coagulation, *J. A. M. A.*, **116:** 578, 1941.
123. White, J. C.: Aneurysms mistaken for hypophyseal tumours, *Clin. Neurosurg.*, **10:** 224, 1964.
124. Wood, E. H.: Angiographic identification of the ruptured lesion in patients with multiple cerebral aneurysms, *J. Neurosurg.*, **21:** 182, 1964.
125. Wright, R. L. and Sweet, W. H.: Carotid or vertebral occlusion in the treatment of intracranial aneurysms; value of early and late readings of carotid and retinal pressures, *Clin. Neurosurg.*, **9:** 163, 1963.

Résumé

Le traitement des anévrismes intracrâniens est un des problèmes majeurs de la neuro-chirurgie actuelle. Un nombre considérable de thérapeutiques a été proposé, et la littérature sur ce sujet comprend plus de 1000 articles que la présente publication résume. La mortalité dans les cas de rupture anévrismale varie entre 50% et 60%; le but de la chirurgie est de stopper le saignement et d'éviter sa récidive, mais peu de chose peut être fait en ce qui concerne les dégâts déjà créés par la compression. En vue d'évaluer les résultats, on peut classer les malades en cinq groupes, selon Botterell: (1) les patients qui sont conscients et porteurs ou non d'un hématome sous-arachnoïdien; (2) ceux qui sont mal orientés, mais ne présentent pas de déficit neurologique; (3) ceux qui sont désorientés et semi-comateux avec un certain degré de déficit neurologique; (4) ceux qui ont un important déficit; (5) ceux qui sont moribonds. Les techniques opératoires qui ont été employées peuvent être groupées en extra-crâniales (ligature de la carotide, occlusion partielle de la carotide, ligature de l'artère vertébrale) et intra-crâniales (ligature de la base de l'anévrisme, oblitération partielle du pédicule anévrismal, stabilisation du sac anévrismal à l'aide de muscle ou de matériels divers, coagulation, excision, ligature d'un vaisseau proche). Récemment on a proposé des interventions visant à rétablir une anatomie vasculaire normale, par "manchonnement" de la lumière du vaisseau intéressé. On a également essayé de thromboser l'anévrisme par voie stéréotaxique. Ces méthodes nouvelles semblent pleines de promesses. L'article se termine par une bibliographie très complète de la question, comportant 125 références.

THE YEAR BOOK OF GENERAL SURGERY (1964-1965 Year Book Series). Edited by M. E. DeBakey. 637 pp. Illust. Year Book Medical Publishers, Inc., Chicago, Ill., 1964. $9.35.

This volume, as in previous years, reviews the significant surgical papers published during the past year. Subjects included are transplantation and artificial organs; shock, fluids and electrolytes; wounds and wound healing; neoplasms; the head and neck; the breast; the thorax and mediastinum; the lungs and pleura; the heart; the aorta and peripheral arteries; peripheral veins; the abdomen—general; the liver and spleen; the biliary tract; the pancreas; the esophagus; the stomach and duodenum; the small intestine and the colon and rectum.

Most of the articles reviewed are from the North American journals with a scattering of reviews from other countries.

The list of "Selected References in Surgery" is excellent, and should be read by every candidate for the Fellowship examinations.

This text provides a ready reference to the current surgical literature and will be of great value to residents in training and to all busy surgeons who do not have the time to read all of the surgical journals.

CASE REPORTS

STOMACH DUPLICATION

JOSEPH P. FLEMING, F.R.C.S.[C],* *Vancouver, B.C.* and
J. N. WARD-McQUAID, M.S., F.R.C.S.,† *Mansfield, England*

DUPLICATION of the stomach is the least common of alimentary tract duplications. The first account of this anomaly was that given by Lyon[5] in 1916. In 1961, Abrami and Dennison[1] described five cases and defined gastric duplications as enteric formations, either spherical or tubular, which lie in contiguity with the normal alimentary tract and share with it a common blood supply and usually a common muscle coat. In a review of 38 cases in 1961, Lewis, Holder and Feldman[4] added one of their own. Other cases have been described by Brodie, Klatzko and Siegman,[2] Nissan,[7] Palmer,[8] Gould and Toffler,[3] and Abrami and Dennison,[1] so in all some 50 cases have been recorded. A review of the various theories concerning alimentary tract duplications was presented by Mousseau, Konno and Hanson[6] in 1963.

CASE REPORTS

CASE 1.—A 15-year-old white girl, a native of Nottinghamshire, was first seen in the pediatric outpatient department of the Mansfield and District General Hospital, Mansfield, England, in 1950. The presenting symptom of central abdominal pain began at 3 years of age, and an episode of melena prompted the pediatric consultation. No cause for these symptoms was found. In 1954 she was admitted to the pediatric department with central abdominal pain, anorexia and melena. The only positive finding was hypochromic microcytic anemia with a hemoglobin of 48%. The hematological findings were otherwise normal. She responded well to therapy with oral iron. A year later, in 1955, she was readmitted with a history of colicky abdominal pain, vomiting and a temperature of 100° F.

Physical examination was negative, and investigations, including an intravenous pyelogram, blood urea and a midstream specimen of urine, were also normal. The pain subsided within 24 hours. Three months later she was admitted with similar abdominal symptoms and constipation. Only two months later a further admission for abdominal pain now accompanied by diarrhea was necessary. A psychoneurotic element in the girl's symptoms was suggested, particularly as they always subsided rapidly after admission. In 1958 she had further abdominal colic which responded to antispasmodics, but in 1960 she was again referred to the pediatric department because of recurrent periumbilical abdominal pain. At this time physical examination was again normal. Investigation included a normal intravenous pyelogram, normal chest radiograph, hemoglobin 71%, leukocyte count 5100/c.mm., erythrocyte sedimentation rate (ESR) 13 mm. in one hour (Westergren). She continued to have intermittent attacks of abdominal pain.

In 1962 at the age of 15 she developed an easily reducible left inguinal hernia and was referred for operative treatment. Apart from the hernia, physical examination was normal. Preoperative investigations included an ESR which was 29 mm. in one hour (Westergren). The hemoglobin was 83% and the blood film showed some anisocytosis. A midstream specimen of urine was normal, and an intradermal test for tuberculosis was negative.

In May 1962 under general anesthesia the left inguinal region was explored. The hernial sac contained an excess of serous peritoneal fluid, some omentum and the left ovary, both of which had surface pigmented patches. A modified Bassini-type repair was performed with silk. Lipofuscin pigment was found in biopsies of the omentum and the left ovary. The peritoneal fluid contained no cells and was sterile.

In the three months following her herniorraphy no physical abnormalities were found but the ESR remained persistently elevated and she had one episode of abdominal pain with diarrhea and vomiting. She was admitted for investigation including a blood film which showed a hemoglobin of 51%, the erythrocytes showing changes of iron deficiency with normal platelets and leukocytes. Three orthotoluidine tests for fecal occult blood were positive. Fecal fat analysis was normal as were other tests for malabsorption. A barium meal and

*1415 - 750 West Broadway, Vancouver, B.C.
†Consultant Surgeon to the Mansfield Group of Hospitals, Mansfield, England.

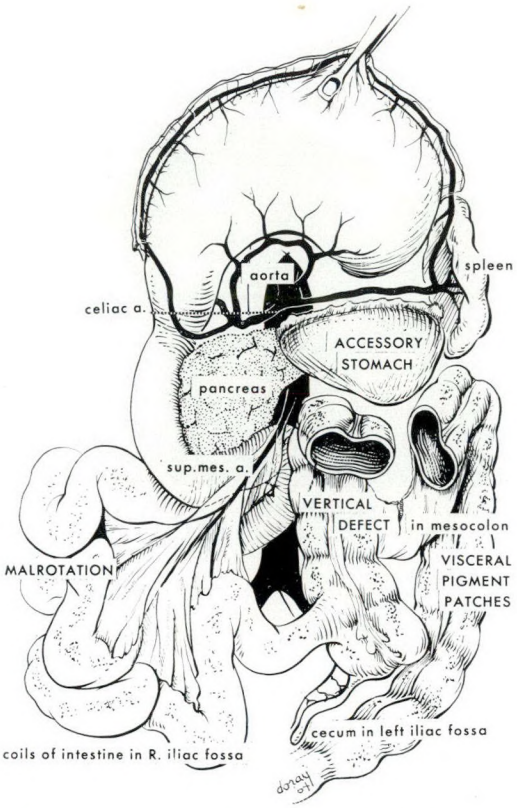


Fig. 1.—Case 1. Drawing of operative findings.

follow-through showed a duodenal ileus which could have been due to malrotation of the duodenojejunal loop.

In view of the persistence of her symptoms, the previous operative findings and the radiographic findings, laparotomy was advised. In November 1962 this was carried out through a right upper paramedian incision under general anesthesia. The main operative findings (Fig. 1) included malrotation of both elements of the midgut and the presence of an accessory stomach. On opening the abdomen, the previously mentioned pigmented areas were seen scattered over the viscera. The cecum was found in the left iliac fossa and the small bowel was confined to the right iliac fossa. The stomach and duodenum were normal to the midline but the duodenum turned here to form the duodenojejunal flexure indicating that the duodenojejunal loop was arrested after 180° of rotation, and the cecocolic loop was arrested after only 90° of rotation. There was also a vertical defect at the midline of the transverse mesocolon which was subsequently repaired. A cystic structure, having the appearance of a distended stomach,

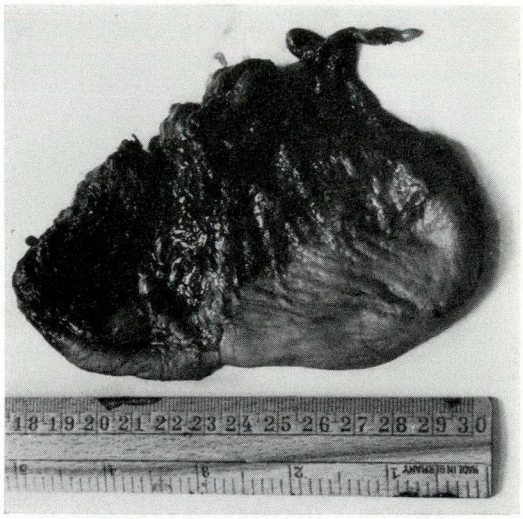


Fig. 2.—Case 1. Photograph of accessory stomach.

was found adjacent to the greater curve of the stomach proper and lying in the gastrocolic omentum. It was adherent to the tail of the pancreas posteriorly and occupied the area between the spleen, stomach and left half of the transverse colon. There was no omentum lying in front of it, but omentum from its "greater curve" ran downwards over the colon. After excision it was found to contain 400 ml. of dark fluid. There was no communication with the lumen of the stomach or adjacent viscera. The duodenojejunal flexure was placed but not fixed in the normal site to the left of the superior mesenteric artery. After an appendectomy, the cecum was similarly placed in the right iliac fossa. Her recovery was uneventful and she was well when seen three months later, and on subsequent follow-up examination her condition was satisfactory.

On gross examination the specimen (Fig. 2) was a structure shaped like a stomach with blackening along one edge. It measured 7 x 12 cm. and the inner surface was lined by two different types of mucosa.

On microscopical examination (Fig. 3), the large gastric-like viscus was lined by a variety of epithelia and mucosa from simple cubical through stratified columnar to a mixture of pyloric and corporeal gastric-like mucosa. There was an area of ulceration which was very suggestive of peptic ulcer. There was an abundant brown pigment apparently the same as that seen in the ovaries and omentum which was lipofuscin.

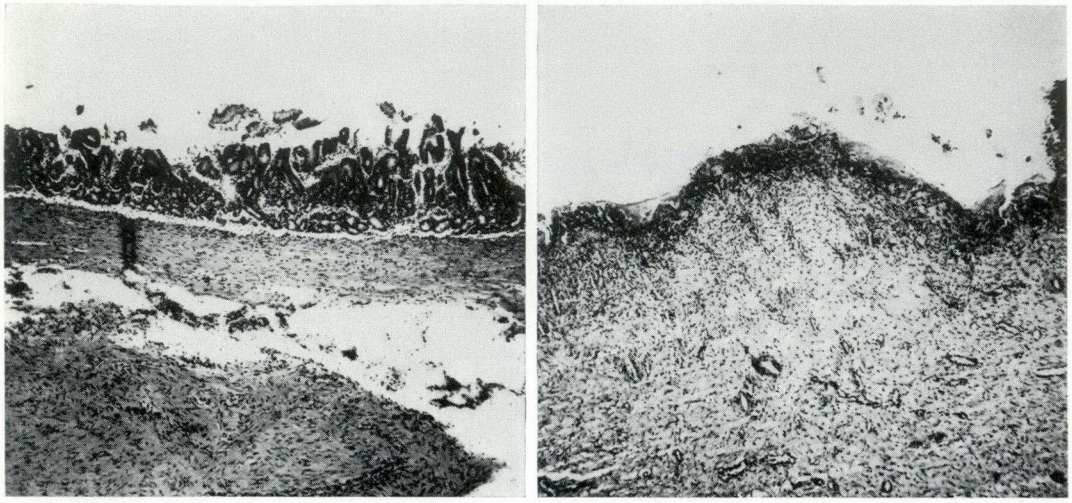


Fig. 3.—Case 1. Composite photomicrograph showing peptic ulceration and mucosa of accessory stomach.

CASE 2.—Baby A.M., a white female born in Vancouver, B.C., was admitted to St. Paul's Hospital on January 14, 1952, at the age of six months. She had a two-day history of nasal discharge and a slight cough associated with moderate dyspnea which led to her admission to hospital. The baby's birth and development were normal and she was breast fed. The family history was non-contributory. Examination revealed a purulent post-nasal drip, decreased breath sounds at the base of the right lung, and a temperature of 100.2° F. The leukocyte count was 18,800/c.mm. with a normal differential; the hemoglobin was 69%. A tentative diagnosis of right lower-lobe pneumonia was made and aqueous penicillin was administered. Films of the chest on the day of admission showed that almost the entire right chest was obscured by a homogeneous density and fluid was present at the right base. The next day a right thoracentesis was carried out and 135 c.c. of chocolate-coloured fluid was aspirated. Subsequent examination showed the fluid to be sterile.

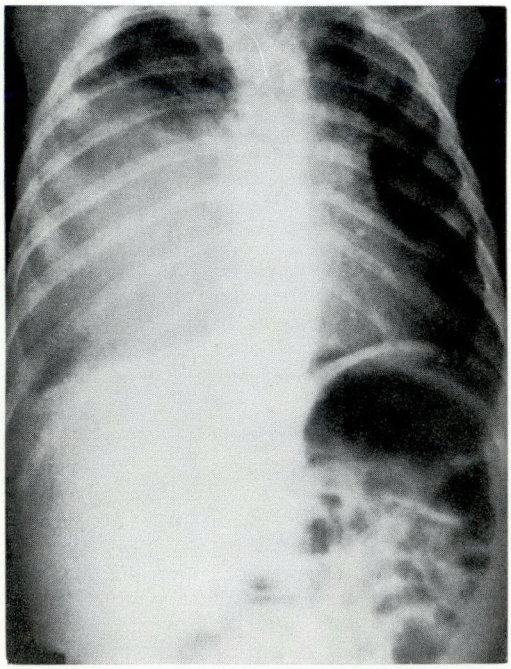


Fig. 4.—Case 2. Chest radiograph.

On January 17, fluoroscopy disclosed a similar homogeneous density of the right chest. A barium swallow was negative for hiatus hernia and there was no mediastinal shift. Films of the spine showed that hemivertebrae were present from cervical vertebra 6 to thoracic 5 inclusive.

The nasal discharge and the respiratory difficulty resolved and the temperature returned to normal within 48 hours of admission. The patient was asymptomatic with a normal leukocyte count one week after admission. On January 25, films of the chest revealed a diminution in the density of the opacification of the right chest and the radiologist reported that the lesion had the appearance of an intrathoracic cyst. The radiological differential diagnosis included dermoid cyst, lung hamartoma or a gastrogenous cyst (Fig. 4).

Since the baby was asymptomatic she was discharged to continue convalescence, and readmission in one month was planned for further investigation and a right thoracotomy.

The patient remained well at home, but on February 13 she developed respiratory dis-

tress and a temperature of 100.6° F.; she was readmitted to St. Paul's Hospital. A complete blood count was normal and penicillin was again administered. The only definite sign present was dullness over the right thorax. Films at this time showed the previously described opacity, but it had enlarged to twice the size it had been three weeks earlier. On February 18, the aspiration of 200 c.c. of clear fluid from the chest relieved the baby's respiratory difficulty. Films after thoracentesis showed a corresponding reduction in the size of the opacity, but one week later the opacity was again its original size. In spite of these radiological findings the child's general condition was satisfactory; she was afebrile and had a normal blood picture. On February 28, 1952, a right thoracotomy was carried out through the fifth intercostal space. The right lung was compressed into the upper chest, but was otherwise normal, and there were no pleural adhesions. An oval cystic structure nearly filled the entire right chest, as the radiograph (Fig. 4) had indicated. The cystic structure was in contiguity with the esophagus, but there was no communication with it. The lesion was excised leaving a completely intact esophagus. A chest drain was inserted and the chest was closed in the routine manner.

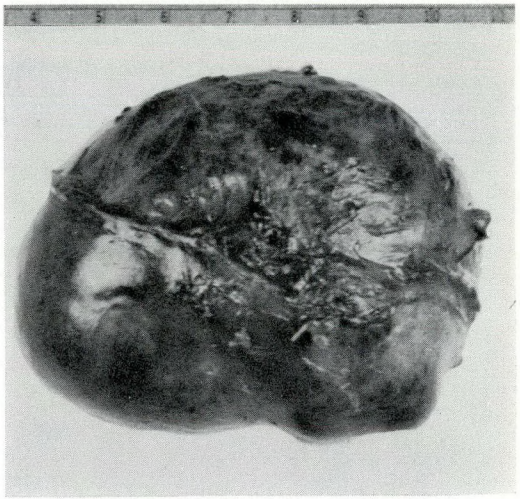


Fig. 5.—Case 2. Photograph of accessory stomach.

On gross examination the specimen (Fig. 5) was an ovoid cystic structure measuring 8 x 6 x 5 cm.; the wall was 0.4 cm. thick and resembled the cut surface of the stomach. The cavity was lined by a velvety tissue and contained thick clear mucoid material. On microscopical examination, multiple sections (Fig. 6) showed a well-defined gastric mucosa

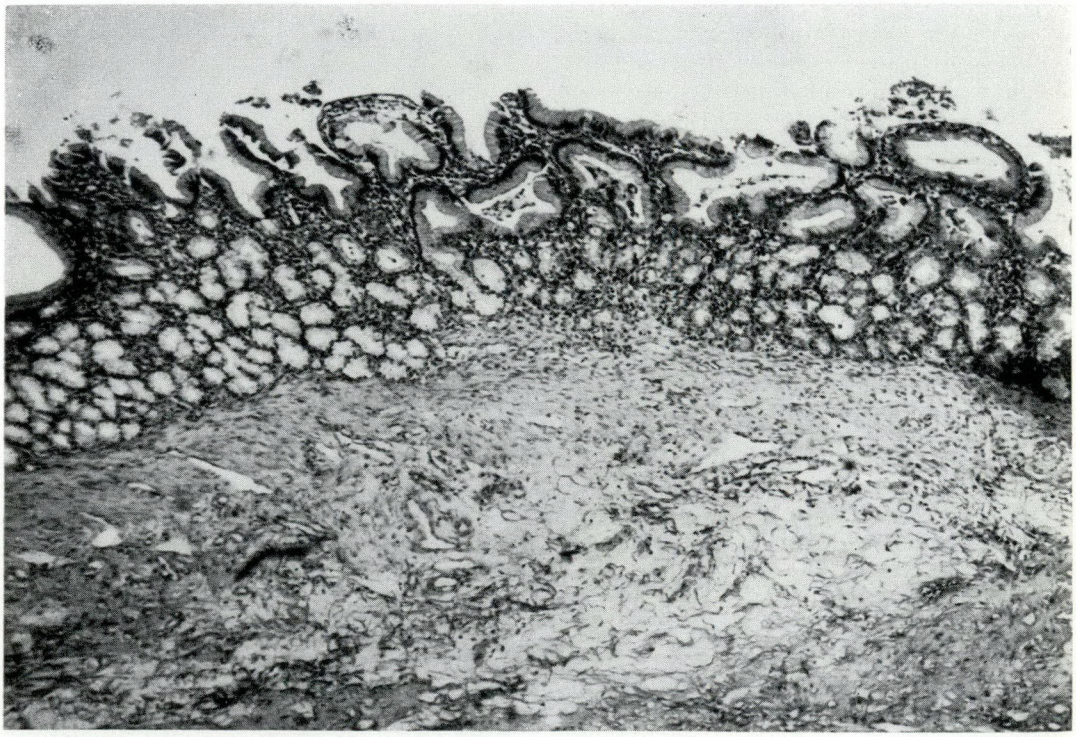


Fig. 6.—Case 2. Photomicrograph of accessory stomach.

and, apart from a few areas of compression, intact columnar epithelium. These cells showed secretory granules. The submucosa was sparsely infiltrated by inflammatory cells, but no frank ulceration was present. There was a well-defined muscularis mucosae and the wall of the structure was composed of two layers of hypertrophied smooth muscle which were arranged in a longitudinal and transverse manner. The fluid content was acidic but no free hydrochloric acid was present.

The patient's postoperative course was uneventful and she was discharged from hospital two weeks after operation. On subsequent follow-up enquiry, the satisfactory immediate postoperative progress had been maintained.

Discussion

The classification of accessory enteric formations and the precise definition of enteric duplication provided by Abrami and Dennison[1] seem the most logical and practical. In only three of the cases previously reported was the duplicated stomach completely separated from the stomach or gut. No other cases of a duplicated stomach with the associated anomaly of intestinal malrotation have been reported, except a case of Abrami and Dennison in which the enteric duplication was not definitely stomach but there was an associated malrotation of the intestines.

The first case presented in this communication illustrates the difficulties encountered in preoperative diagnosis in gastric duplication; these have been emphasized by Abrami and Dennison. The diagnosis was further obscured in this case because of the associated anomaly of malrotation of the gut. The nonspecific nature and chronicity of the symptoms in this youngster made a final diagnosis of psychoneurosis very tempting. If it were not for the findings of lipofuscin and free fluid at the time of herniorrhaphy, subsequent laparotomy may not have been undertaken. The symptomatology of gastric duplication may mimic peptic ulcer and its complications, including pyloric stenosis, bleeding and perforation. Volvulus of the malrotated gut may also produce obstructive symptoms with melena and hematemesis. The periodic syndrome and mesenteric adenitis may produce similar symptoms. The symptoms present over the years in the first case could have resulted from any one of these conditions. The presence of the pigment, lipofuscin, both in the accessory stomach and the peritoneal cavity suggests that this pigment may have escaped from the accessory stomach during previous episodes of ulceration and perforation. The presence of a peptic ulcer in the accessory stomach is also sufficient to explain the intermittent attacks of abdominal pain. Gould and Toffler[3] suggest that barium studies may offer a clue to intestinal duplication when a smooth-walled pressure defect is seen anywhere along the greater curve, particularly in the antral region. If a communication exists between the main stomach and the accessory stomach, barium may outline the duplication. In Case 1, radiological evidence suggesting duodenal ileus can be accounted for by the malrotated duodenojejunal loop. All writers agree that surgical excision of the gastric duplication is the treatment of choice and may consist of simple excision (as in Case 1) or partial gastrectomy. Nissan[7] mentions other operative procedures including destruction of the mucosa with external drainage or marsupialization. In the first case, the performance of jejunoduodenostomy was considered as treatment of the malrotated duodenojejunal flexure, but since all of the symptoms were probably produced by the accessory stomach, the intestines were placed in their normal anatomical position without fixation. The incidental appendectomy may contribute to the fixation of the repositioned cecum in the right iliac fossa. The defect in the transverse mesocolon was repaired and obliterated in order to prevent an internal hernia.

In Case 2, the radiographic features, which indicated a cystic lesion in the chest, and the associated anomaly of hemivertebrae should suggest the possibility of stomach duplication.

Summary

Two cases of gastric duplication are reported. The first is that of a 15-year-old white girl, a native of Nottinghamshire, England, in whom there was a completely

separate duplicated stomach and intestinal malrotation. Diagnostic features included the operative finding of free fluid with lipofuscin pigment in a hernial sac, which led to the definitive surgical treatment.

The second case is that of a white female infant born in Vancouver, Canada. In contrast to the first case in whom chronicity of symptoms was an outstanding feature, this seven-month-old infant had definitive surgical treatment within six weeks of the onset of her symptoms. Earlier operation in this case is attributed to the intrathoracic location of the accessory stomach and the associated respiratory symptoms.

In the preparation of the first case, the authors are indebted to Dr. A. MacFarlane, Department of Pathology, for his histological reports, and to Dr. A. C. Blandy, Department of Pediatrics, and Dr. J. P. Caley, Department of Medicine, for their records at the Mansfield and District General Hospital, Mansfield, England. One of us (J.P.F.) is indebted to Dr. Robert H. Gourlay for permission to include his case of intrathoracic gastric duplication, and for his advice in preparing these reports. Our thanks also are extended to Dr. H. H. Pitts, Sr., and Dr. John H. Sturdy, St. Paul's Hospital, Vancouver, for their pathology reports.

References

1. Abrami, G. and Dennison, W. M.: Duplication of the stomach, *Surgery*, **49:** 794, 1961.
2. Brodie, N., Klatzko, M. and Siegman, F. A.: Duplication of the stomach associated with bleeding duodenal ulcer, *A.M.A. Arch. Surg.*, **80:** 354, 1960.
3. Gould, R. J. and Toffler, A. H.: Duplication of the stomach; a case report, *Radiology*, **76:** 790, 1961.
4. Lewis, P. L., Holder, T. and Feldman, M.: Duplication of the stomach: report of a case and review of the English literature, *A.M.A. Arch. Surg.*, **82:** 634, 1961.
5. Lyon, W.: A case of congenital cyst of stomach, *J. Mich. Med. Soc.*, **15:** 42, 1916.
6. Mousseau, L. P., Konno, T. and Hanson, S.: Duplication of the rectum, *Canad. J. Surg.*, **6:** 438, 1963.
7. Nissan, S.: Duplications of the stomach, *Amer. J. Surg.*, **100:** 59, 1960.
8. Palmer, T. H., Jr.: Duplication of the stomach, *J. Maine Med. Ass.*, **51:** 424, 1960.

Résumé

Les dédoublements de l'estomac constituent le type le plus rare de dédoublement le long du tractus digestif. Le premier cas que l'on trouve dans la bibliographie remonte à 1916. Au total, il en a été décrit depuis environ 50 autres. Les auteurs présentent ici l'histoire résumée de deux malades. La première est celle d'une jeune fille de 15 ans dont les troubles remontaient à 8 ans auparavant. Elle avait déjà subi plusieurs hospitalisations pour des douleurs abdominales, d'anorexie, de méléna et de vomissements. Un grand nombre d'examens avaient été pratiqués ici et là, mais le diagnostic n'avait pu être fait et des troubles psychiques avaient même été envisagés comme probables. En 1962, elle est opérée pour une hernie inguinale gauche, ce qui se passe sans histoire particulière. Quelques mois plus tard, les symptômes abdominaux réapparaissent et cette fois-ci on détecte un trouble du transit baryté au niveau du duodénum, suggérant un iléus. On procède alors à une laparotomie qui permet de découvrir une rotation incomplète des deux éléments de l'intestin moyen associée à l'existence d'un estomac supplémentaire; le cæcum est logé dans la fosse iliaque gauche. L'estomac accessoire est adhérent au pancréas et communique avec l'estomac normal, et l'on en fait l'excision. Les anses intestinales déplacées sont remises, mais non fixées, à leur place normale. Les suites opératoires furent excellentes et la malade est maintenant parfaitement guérie de ses troubles. Le second cas est celui d'une petite fille de six mois, admise à l'hôpital pour un épisode de toux avec température élevée. A l'auscultation, il existe une diminution des bruits respiratoires au niveau de la base pulmonaire droite et l'on pose le diagnostic de pneumonie de la base droite. Les examens radiographiques suggèrent la possibilité d'une tumeur kystique. Une thoracotomie est effectuée quelques semaines plus tard et l'on trouve effectivement une masse kystique adhérente à l'œsophage, mais ne communiquant pas avec lui. Cette lésion est facilement excisée, sans que la paroi œsophagienne soit même blessée. L'examen anatomopathologique révèle que ce kyste est tapissé d'une muqueuse de type gastrique et l'on se trouve donc en présence d'un estomac rudimentaire, mais possédant cependant sa muqueuse et ses muscles. Les suites opératoires furent sans histoire. Ce n'est que dans trois cas, rapportés dans la littérature, que l'on trouve un estomac accessoire totalement détaché du tractus digestif.

ACUTE ALCOHOLIC POISONING: A COMPLICATION OF GASTRIC HYPOTHERMIA*

BERNARD J. F. PEREY, M.D., F.R.C.S.[C],† STEPHEN J. HELLE, M.D.‡ and LLOYD D. MacLEAN, M.D., Ph.D.,§ *Montreal, Que.*

Gastric hypothermia for the control of severe upper gastrointestinal hemorrhage, a relatively new technique, has become widely popular within the last three years.[1-3] In most cases, a gastric hypothermia machine is used to circulate a solution of ethyl alcohol through a balloon which has been introduced into the stomach. Rupture of the balloon with release of large quantities of alcohol is a potential hazard. In the case to be described, a potentially lethal quantity of alcohol was released when the gastric balloon became disconnected from the tubing.

Case Report

S.K., a 72-year-old man, was admitted to hospital on November 11, 1963, with an eight-hour history of melena and hematemesis. Past history disclosed a similar episode requiring admission and multiple blood transfusions in 1955. The blood pressure on admission was 90/70 mm. Hg, pulse 128/min. and hemoglobin 13.5 g. %. An emergency barium meal carried out on admission failed to reveal the source of bleeding. The presumptive diagnosis at that time was bleeding duodenal ulcer.

During the first 24 hours after admission, the patient received nine bottles of blood in order to keep the systolic blood pressure above 100 mm. Hg. In spite of this, the hemoglobin fell to 9.5 g. %. The central venous pressure remained below 1 cm. of water.

In view of the failure of more conservative therapy, gastric cooling was instituted 24 hours after admission. After removal of intragastric blood clots by gastric lavage and following topical anesthesia of the throat, a gastric balloon was passed into the stomach and was filled with 1000 ml. of absolute ethyl alcohol. The coolant was circulated through the balloon with the Swenko gastric hypothermia machine at a return-flow temperature of approximately 7° C. Five bottles of blood were given and the systolic blood pressure was stabilized at 130 mm. Hg.

After 12 hours of cooling, i.e. at 8:00 a.m. on November 13, the patient became restless and confused and before he could be restrained pulled vigorously on the plastic tube leading to the balloon. The tube became disconnected from the balloon which remained in the stomach. The patient promptly vomited several hundred millilitres of blood-tinged alcohol. Gastric lavage with 2 litres of normal saline was performed within 20 minutes of the accident. Because the patient began to cough copious amounts of blood-tinged foamy sputum and coarse rales were heard throughout both lung fields, aspiration of vomitus was believed to have taken place. A tracheostomy was performed immediately, followed by lavage of the tracheobronchial tree with 500 ml. of normal saline. Hydrocortisone, 100 mg., was administered intravenously.

Two hours after the accident, the patient showed no gross evidence of alcoholic intoxication and was able to communicate intelligently. The blood alcohol level was 100 mg. %. Nevertheless, hemodialysis was decided upon because a certain amount of alcohol may have remained trapped in the gastric balloon and could at any time pour into the stomach and cause delayed alcoholic poisoning. Indeed, upon transfer to the dialysing unit 2½ hours after the accident, the blood pressure fell from 130/80 to 90/50 mm. Hg, the pulse rate rose to 140/min., and the patient began to lose consciousness. Hemodialysis was instituted about three hours after the accident. Twenty minutes after dialysis had begun, he responded only to the sharp pain of supraorbital pressure and was completely areflexic. Blood pressure and pulse rate had returned to normal without blood transfusions. Blood alcohol levels were not determined at this time. After 85 minutes of dialysis, the level of consciousness had definitely improved and response to pain was easily elicited. After 100 minutes of dialysis, he responded to the spoken word and, at 120 minutes, showed no signs of mental confusion. The procedure was maintained for a total of four hours. No transfu-

*From the Departments of Surgery and Medicine, Royal Victoria Hospital and McGill University, Montreal.

Supported by the Medical Research Council of Canada, Grant No. MA-1413.

†Assistant Surgeon, Royal Victoria Hospital; Lecturer in Surgery, McGill University.

‡Assistant in the Outdoor Clinics, Department of Medicine, Royal Victoria Hospital.

§Surgeon-in-Chief, Royal Victoria Hospital; Professor of Surgery, McGill University.

sions were given during dialysis. Corticosteroids, vitamin B_1 and antibiotics were administered during and after dialysis.

Twelve hours after the accident, as internal bleeding appeared to continue, a laparotomy was performed. The gastric mucosa was pale, thickened and edematous and approximately 20 circular erosions were noted throughout the body and antrum, each measuring about 5 mm. in diameter. Diffuse oozing of blood was present but no major site of bleeding could be found. There was no duodenal bleeding or ulceration. The gastric balloon, which was empty, was removed and a vagotomy and pyloroplasty were performed.

The postoperative course was complicated by prolonged paralytic ileus, bronchopneumonia and two dehiscences of the wound which required operative closure. The patient recovered and was discharged from hospital on January 15, 1964.

Discussion

Two noteworthy aspects of this case are (1) the serious, and previously unreported, complication of alcoholic intoxication following gastric hypothermia, and (2) successful treatment by hemodialysis.

This accident would have been of little consequence if the coolant had been nontoxic. While pure ethyl alcohol is the required coolant for gastric freezing with the Swenko machine, a 50% dilution is recommended for gastric cooling. Even lower concentrations of alcohol should be adequate with this equipment. Non-toxic synthetic coolants such as silicone derivatives are being investigated in Wangensteen's laboratory.[4] Propylene glycol is the nontoxic coolant used in the Weck Cryocycle machine.* These coolants are somewhat viscous and require specially designed apparatus. Other investigators have developed simple devices, making use of ice-cooled tap water as a coolant.[5, 6]

The fact that this patient survived the administration of a lethal dose (1000 c.c.) of ethyl alcohol appears to be due to several factors. High concentrations of alcohol are not tolerated by the stomach and cause immediate vomiting. Much of the alcohol was removed by vomiting and gastric lavage. The clinical picture of rapid deterioration, followed by a dramatic and equally rapid recovery, would indicate that hemodialysis played an important role in reducing dangerously high blood alcohol levels.

Alcohol is usually rapidly absorbed from the stomach and small intestine and peak blood levels are reached in man about 60 minutes after alcoholic ingestion.[7] However, in our patient, the clinical peak of intoxication, as judged by the level of consciousness, was about 3.5 hours after the accident. A possible explanation for this delay is that while most of the alcohol lying free in the stomach shortly after the accident was probably removed by vomiting and gastric lavage, a certain amount may have been retained in the gastric balloon and spilled at a later time, such as during the transfer of the patient to the dialyzing unit.

The value of hemodialysis in massive ethanol poisoning has been demonstrated experimentally in dogs by Marc-Aurele and Schreiner,[8] although the method apparently has never been used clinically. The duration of dialysis needed by our patient before he showed a clinical response is in keeping with the experience of these authors. While their control dogs remained unconscious for at least 10 hours, the dialyzed dogs were awake by the second hour of the procedure.

Correlation between blood levels of alcohol and clinical behaviour is reasonably well known.[7] Only 25% of persons with blood levels of 100 mg. % have some clinical evidence of intoxication. Severe intoxication is present with levels of 400 mg. %, while levels of 500 to 800 mg. % are usually fatal.

It is not known how much the edematous ulcerated appearance of the gastric mucosa was due to its exposure to alcohol. These changes may have been present before cooling, representing a severe hemorrhagic gastritis. It is not likely that gastric cooling by itself produced such changes. It has been our experience that patients who have undergone prolonged gastric cooling do not necessarily display any significant gastric mucosal change when examined at gastroscopy or laparotomy.

*Edward Weck & Co., Long Island City, New York.

Conclusions

Ethanol in high concentrations is not a safe coolant to use for gastric cooling. Newer devices making use of non-toxic coolants prevent serious consequences in the event of accidental spillage of the material into the stomach. However, the presence of a large quantity of alcohol in the stomach does not necessarily cause death. Hemodialysis has proved to be a dramatically efficient way to treat acute massive ethanol poisoning. It is suggested that all centres using alcohol as a coolant for gastric hypothermia maintain gastric lavage instruments, suction apparatus and hemodialysis facilities readily available.

Summary

A case of acute massive ethanol poisoning complicating gastric cooling for hemorrhage is presented. The use of gastric lavage, tracheostomy and hemodialysis resulted in survival of the patient. The value of hemodialysis and the lessons learned from this case are discussed.

We are indebted to Dr. G. Joron of the Montreal General Hospital for the blood alcohol determination.

References

1. Nicoloff, D. M. *et al.*: Local gastric hypothermia in the management of massive gastrointestinal hemorrhage, *Surg. Gynec. Obstet.*, **114:** 495, 1962.
2. Wangensteen, S. L.: Intragastric cooling for upper gastrointestinal hemorrhage, *Surg. Clin. N. Amer.*, **42:** 1171, 1962.
3. Miller, R. E., Moscarella, A. A. and Fitzpatrick, H. F.: Local gastric hypothermia, *Arch. Surg. (Chicago)*, **86:** 272, 1963.
4. Goodale, R.: Personal communication.
5. Small, M. D. and Brick, I. B.: Simple method for achieving gastric cooling, *J. A. M. A.*, **187:** 678, 1964.
6. Holt, M. H.: A simple cooling device for treatment of upper gastrointestinal bleeding, *Surgery*, **50:** 728, 1961.
7. Goodman, L. S. and Gilman, A.: The pharmacological basis of therapeutics: a textbook of pharmacology, toxicology, and therapeutics for physicians and medical students, 2nd ed., The Macmillan Company, New York, 1955.
8. Marc-Aurele, J. and Schreiner, G. E.: The dialysance of ethanol and methanol: a proposed method for the treatment of massive intoxication by ethyl or methyl alcohol, *J. Clin. Invest.*, **39:** 802, 1960.

Résumé

Histoire d'un cas.—Un homme de 72 ans est admis à l'hôpital en novembre 1963 pour méléna et hématémèse remontant à huit heures auparavant. Le diagnostic d'entrée est "hémorragie d'un ulcère duodénal". Des transfusions abondantes sont faites pendant les 24 premières heures, sans amélioration. On pratique alors un refroidissement gastrique: sous anesthésie locale de l'arrière-gorge, on passe un ballon où l'on injecte un litre d'alcool éthylique absolu que l'on refrigère. Un peu plus tard, lors d'une crise d'excitation, le malade tente d'arracher le tube œsophagien et de cette façon, il détache l'insertion du ballon, ce dernier restant dans l'estomac; on assiste alors à une intoxication progressive par alcool, que l'on traite par hémodyalise. L'alcool sanguin est titré au laboratoire. Malgré une période transitoire de perte de conscience totale, le malade a pu être ranimé; le lendemain, on effectue une laparotomie et une gastrotomie: il est impossible de trouver une origine précise au saignement. La muqueuse gastrique porte sur toute sa surface de petits ulcères et l'on fait une vagotomie associée à une pyloroplastie. Les suites opératoires furent compliquées d'un iléus et de bronchopneumonie; cependant le malade finit par guérir et put être renvoyé à domicile au milieu de janvier 1964. L'intérêt de ce cas est de souligner un danger inattendu des techniques de refroidissement gastrique: l'intoxication alcoolique aiguë. Il semble qu'il y aurait un très net intérêt à utiliser un réfrigérant non toxique. On a récemment proposé de diluer l'alcool à moins de 50%, de le remplacer par du propylène-glycol ou des dérivés siliconés.

CORRECTION

It is very much regretted that because of typographical errors the names of Drs. Jules Charron and François Telmosse were incorrectly spelled on page 23 of the January 1965 issue of the Journal.

RUPTURE OF THE INTERVENTRICULAR SEPTUM DUE TO BLUNT TRAUMA*

M. TASCON-ALONSO, M.D., M.Sc. and O. ROSTRUP, M.D., F.R.C.S.[C],
Edmonton, Alta.

WHILE rupture of the heart wall from nonpenetrating injury is not uncommon, rupture of the interventricular septum is a rare complication of trauma to the chest. This injury is most frequently seen in young persons who have suffered blunt trauma from the steering wheel in automobile accidents. Such an occurrence in a 21-year-old man is described in the present communication.

CASE REPORT

A 21-year-old white man was admitted to the University of Alberta Hospital on September 29, 1963, having been transferred from an outlying hospital to where he had been taken following a car accident on September 28, 1963. On admission he was unconscious and was suffering from traumatic shock, chest contusions, multiple lacerations and fractures of the lower limbs. His chest injury was presumed to be due to violent contact with the steering wheel. Before the accident, he had been active and in good health.

On physical examination on the day of admission to the University Hospital he was moderately confused at times, but responded rationally to commands. His injuries included multiple abrasions of the face, lacerations of the upper and lower extremities and contusions of the head and chest. The heart sounds were distant. A Grade III (Grades I-IV) rough apical presystolic and short diastolic blowing murmurs were heard over the entire precordium. The blood pressure was 85/40 mm. Hg and pulse rate was 140 per minute. Respiratory rate was 30 per minute. There was slight tenderness over the left pubic area and hematuria was noted. His temperature was 99.5° F. No peripheral edema or cyanosis was observed.

Roentgenograms on admission showed fractures of the neck of the left femur, right acetabulum and ischiopubic ramus, shaft of the right and left femur, left tibia and fibula and right ankle. In addition, there was a fracture of the fifth left rib in the mid-axillary line (Fig. 1). The heart was normal in size; the pleural cavities and the lung fields were normal. A cystogram revealed no evidence of damage to the bladder.

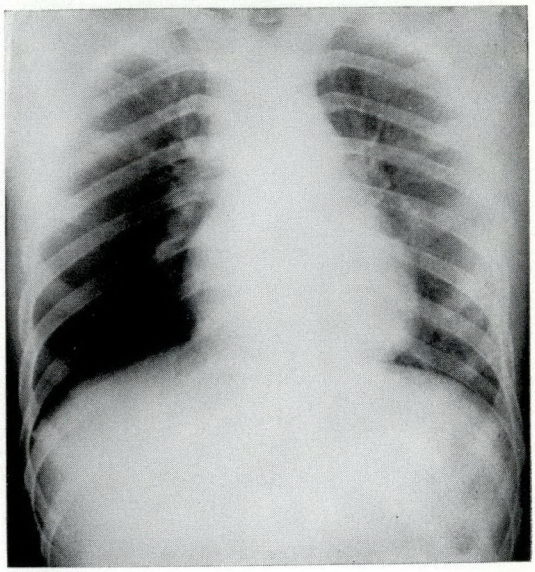


Fig. 1.—Roentgenogram of the chest taken on admission.

The leukocyte count was 20,000/c.mm. with a normal differential count, and the hemoglobin was 11.3 g./100 c.c. A urine specimen contained numerous erythrocytes.

He was treated with blood transfusions, skin traction and Thomas splints to both lower extremities. On September 30, 1963, (36 hours after his accident) he was taken to the operating room where plaster casts were applied to both legs. The patient's vital signs remained stable for the rest of the day with a blood pressure of 120/55 mm. Hg and a pulse rate of 100 per minute. At 2 a.m. on October 1, 1963, the patient became restless and confused. A second radiograph of the chest showed congestion of both lungs. His blood pressure and pulse remained unchanged and his extremities were warm and of good colour. After the administration of oxygen and meperidine hydrochloride the patient slept for short periods. By 5 a.m. that morning he developed increased orthopnea and peripheral cyanosis. The plasma CO_2 content at this time was 15.3 mEq./l. He died suddenly at 7 a.m. on October 1, 42 hours after admission.

*Orthopedic Department, University of Alberta Hospital, Edmonton.

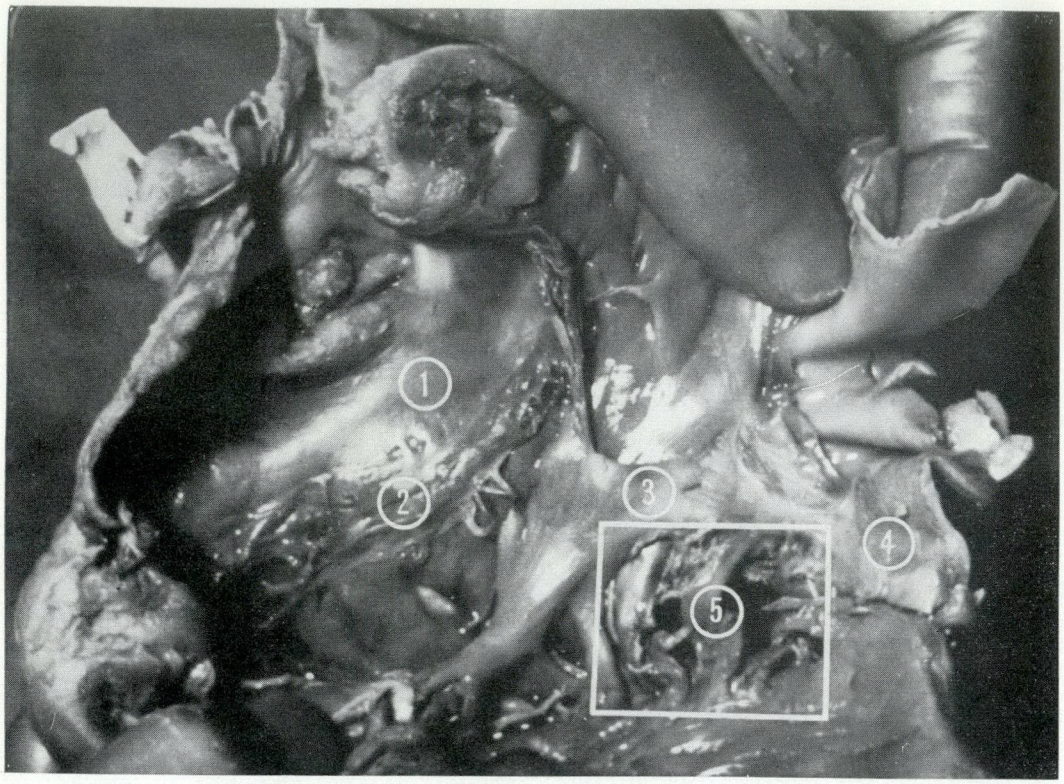
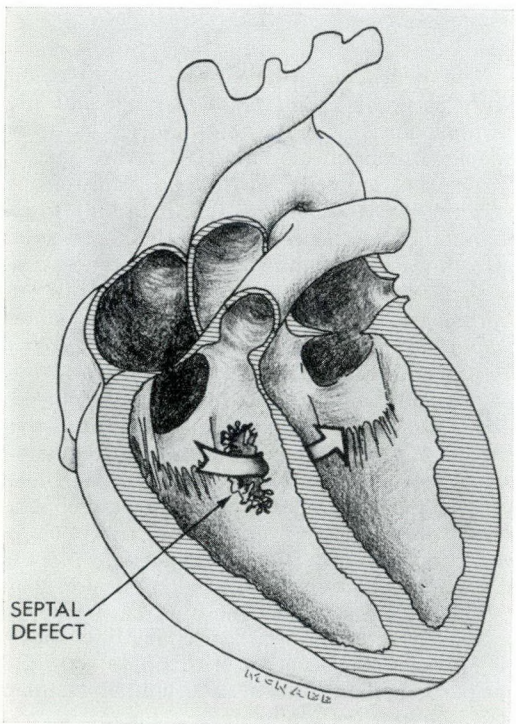


Fig. 2.—1. Right atrium. 2. Septal leaflet tricuspid valve. 3. Supraventricular crest. 4. Pulmonary valve cusps. 5. Ventricular septum demonstrating laceration.



(Left) **Fig. 3.**—The anatomical position and size of the ventricular septal defect.

The discovery of a contused chest, a presystolic and short diastolic murmurs, a resultant heart failure and rather sudden death, 55 hours after the accident, led to the clinical diagnosis of a traumatic septal defect, which was later confirmed at autopsy.

Autopsy.—Post-mortem examination was performed at 3 p.m. on the same day and confirmed the presence of the fractures and lacerations already described. Both pleural cavities contained blood; the left, 125 ml., and the right, 105 ml. By gross and microscopical examination, both lungs and liver showed marked congestion and edema. There were superficial lacerations of the spleen. The heart weighed 450 g. There was a laceration of the pericardium along its left lateral border about 10 cm. in length. No blood was noted in the pericardial cavity. The atria and ventricles were unremarkable. The aorta, coronary arteries and peripheral vessels were intact. The valves were normal. There was a laceration of the interventricular septum mea-

suring 1.5 to 2 cm. in diameter (Figs. 2 and 3).

Sections through the region of the tear in the interventricular septum showed necrosis and acute inflammatory reaction. Some distance from the laceration there was an area of hemorrhage into the myocardium. No evidence of brain injury was found.

Discussion

In 1847, Hewett[9] reported five cases of rupture of the heart and large vessels as a result of injury. In 1952, Pollock, Markelz and Shuey[14] reviewed the literature concerning blunt trauma to the heart and recorded 12 instances in which rupture of the interventricular septum occurred. Guilfoil and Doyle[8] in 1953 described the first patient in whom the diagnosis was established by cardiac catheterization.

Automobile accidents and crush injuries to the chest are usual causes of cardiac trauma. The dynamics of such injuries may be influenced by the phase of the cardiac cycle at the moment of injury, as well as the direction of chest compression.[2, 12]

Lacerations may occur immediately or may be delayed, resulting from perforation of necrotic contused myocardium. Most cases have been reported in young men and, in most of them, a systolic murmur and thrill have been noted in the fourth and fifth left intercostal spaces. In a few cases, no murmurs or thrills were heard initially, perhaps as a result of hypotension. The presence of an interventricular septal laceration in cases of severe trauma may not be recognized when the patients die shortly after injury. Of 18 patients reviewed by Peirce, Dabbs and Rawson,[13] 10 died within 15 days of congestive heart failure. Some patients, however, have survived as long as 11 years. Successful closure of acquired septal defects with the use of extracorporeal circulation have been reported.[3, 5, 7, 13, 17, 18]

Summary

A healthy 21-year-old man sustained a rupture of the interventricular septum in an automobile accident. He died three days after the accident from rapid progressive cardiorespiratory deterioration. This experience emphasizes the need to consider this complication in patients who have suffered chest trauma. Surgical repair of the defect is possible and must be considered as an emergency procedure in patients who develop cardiac failure after an accident of this nature.

We wish to thank Drs. J. C. Callaghan and R. S. Fraser for their advice and criticism during the preparation of this paper.

References

1. Arenberg, H.: Traumatic heart disease: clinical study of 250 cases of non-penetrating chest injuries and their relation to cardiac disability, *Ann. Intern. Med.*, **19:** 326, 1943.
2. Bright, E. F. and Beck, C. S.: Nonpenetrating wounds of the heart; clinical and experimental study, *Amer. Heart J.*, **10:** 293, 1935.
3. Campbell, G. S. *et al.*: Traumatic ventricular septal defect; report of two cases, *J. Thorac. Surg.*, **37:** 496, 1959.
4. Carey, F. H., Hurst, J. W. and Arentzen, W. R.: Acquired interventricular septal defect secondary to trauma, *New Eng. J. Med.*, **258:** 355, 1958.
5. Cleland, W. P. *et al.*: Repair of ventricular septal defect following indirect trauma, *Brit. J. Dis. Chest*, **55:** 17, 1961.
6. Paulin, C. and Rubin, I. L.: Complete heart block with perforated interventricular septum following contusion of chest, *Amer. Heart J.*, **52:** 940, 1956.
7. Desforges, G. and Abelmann, W. H.: Interventricular septal defect due to blunt trauma; report of a case repaired surgically under total cardiopulmonary bypass, *New Eng. J. Med.*, **268:** 128, 1963.
8. Guilfoil, P. H. and Doyle, J. T.: Traumatic cardiac septal defect: report of a case in which diagnosis is established by cardiac catheterization, *J. Thorac. Surg.*, **26:** 510, 1953.
9. Hewett, P. G.: Cases of rupture of the heart and large vessels; the results of injuries, *Trans. Path. Soc. Lond.*, **1:** 81, 1846-8.
10. Leonard, J. J., Harvey, W. P. and Hufnagel, C. A.: Rupture of aortic valve; therapeutic approach, *New Eng. J. Med.*, **252:** 208, 1955.
11. Mahaffey, D. E., Schramel, R. and Creech, O., Jr.: Traumatic ventricular septal defect: report of a case treated successfully, *J. Louisiana Med. Soc.*, **109:** 321, 1957.
12. Moritz, A. R. and Atkins, J. P.: Cardiac contusion; experimental and pathologic study, *Arch. Path. (Chicago)*, **25:** 445, 1938.
13. Peirce, E. C., II, Dabbs, C. H. and Rawson, F. L.: Isolated rupture of ventricular septum due to non penetrating trauma, *A.M.A. Arch. Surg.*, **77:** 87, 1958.
14. Pollock, B. E., Markelz, R. A. and Shuey, H. E.: Isolated traumatic rupture of interventricular septum due to blunt force, *Amer. Heart J.*, **43:** 273, 1952.
15. Rubenstein, P. and Levinson, D. C.: Acquired interventricular septal defects due to myocardial infarction and nonpenetrat-

ing trauma to the chest, *Amer. J. Cardiol.*, **7:** 277, 1961.

16. Stern, W. R. and Stoddard, L. D.: Traumatic interventricular septal defect of the heart; a case report, *Amer. Heart J.*, **63:** 821, 1962.
17. Therkelsen, F.: Surgical repair of traumatic ventricular septal defects, *Acta Chir. Scand.*, **119:** 372, 1960.
18. Trias, M. and Vejarano, A.: Successful suturing of a traumatic interventricular communication, *Angiologia*, **15:** 98, 1963.

Résumé

La rupture du cœur à la suite d'un traumatisme non pénétrant est une complication relativement fréquente. Par contre, la rupture isolée du septum interventriculaire est très rare. Les auteurs rapportent ici un cas de ce genre. C'est celui d'un homme de 21 ans, admis à l'Hôpital Universitaire de l'Alberta en septembre 1963, pour un accident d'automobile. Le malade était dans un état de choc grave, inconscient et présentait une contusion du thorax ainsi que de multiples fractures et lacérations des membres inférieurs. A l'auscultation, on découvre un souffle cardiaque couvrant toute la région précordiale. Les radiographies ne montrent rien d'anormal à ce niveau, mais de nombreuses fractures des deux membres inférieurs, et de la cinquième côte gauche. Le pouls est à 140 et la pression à 85/40. Le traitement est d'abord conservateur: transfusion et réanimation. Trente-six heures plus tard, l'état général s'améliore légèrement et l'on fait des plâtres aux deux jambes. Le lendemain, le malade devient brusquement inconscient et meurt soudainement. Une autopsie est pratiquée. Les deux cavités pleurales contiennent du liquide sanglant. Il existe une déchirure du péricarde au niveau de son bord externe gauche: extérieurement, le cœur semble normal, mais il est trouvé une déchirure de la paroi interventriculaire mesurant 2 cm. Cette lésion était opératoirement curable. La difficulté d'en poser le diagnostic clinique est très grande.

TRAITEMENT CHIRURGICAL DES PROLAPSUS GENITAUX. R. Merger, J. Lévy, J. Melchior and J. Barrat. 74 pp. Illust. Masson & Cie, Paris, 1964. 26F. $5.20 (approx.).

Volume très bien illustré qui représente une critique et une analyse du traitement chirurgical des prolapsus génitaux en France. L'étude couvre une période de 30 ans.

Après une très bonne description d'une technique type et de ses modifications suivant les cas donnés les auteurs déterminent la technique de leur choix.

En terminant, les auteurs énumèrent les indications, les soins post-opératoires, les complications et les récidives.

Une étude statistique complète le travail. Cet ouvrage n'ajoute rien aux notions déjà connues du traitement chirurgical des prolapsus génitaux.

RUPTURES TRAUMATIQUES DES BRONCHES ET DE LA TRACHEE THORACIQUE. J. Dor, E. Forster and H. Le Brigand. 230 pp. Illust. Editions Doin, Deren & Cie, Paris, 1964. 54F. $10.80 (approx.).

Les auteurs concluent que le pronostic des ruptures traumatiques des bronches et de la trachée thoracique repose sur la précocité du diagnostic suivi d'une réparation immédiate.

Se basant sur une analyse de 264 observations cliniques, voici les notions actuelles sur ce problème:

Selon eux si le mécanisme et la pathogénie des ruptures trachéales et bronchiques restent encore obscures sur bien des points, l'évolution anatomique des lésions et le retentissement physiopathologique par contre sont bien connus. La connaissance précise de cette physiopathologie précoce nous permet d'intervenir et de rééquilibrer le grand traumatisme thoracique.

De l'analyse clinique le praticien doit garder à l'esprit trois notions: Le syndrome gazeux compressif doit faire évoquer automatiquement l'hypothèse d'une rupture trachéo-bronchique, tout syndrome gazeux ne doit pas être négligé, seule la bronchoscopie peut confirmer le diagnostic et orienter vers un traitement chirurgical adéquat.

Le diagnostic étant affirmé, le succès thérapeutique dépend de trois points: réparation précoce, intervenir sur un patient préalablement rééquilibré et bien compensé, ne pas négliger l'existence possible d'autres lésions traumatiques, celles du névraxe en particulier.

Quand les circonstances auront voulu que la réparation ne puisse pas être immédiate ou précoce, quelques notions peuvent encore servir de guide. Il vaut mieux éviter d'intervenir en phase retardée ou au début de la période secondaire. Il vaut mieux ne pas attendre trop longtemps non plus, pour éviter une détérioration lente de l'architecture pulmonaire, en dehors même de toute infection. Il vaut mieux chercher à faire une intervention réparatrice plutôt qu'une exérèse.

Il faut espérer que les cas de ruptures trachéales ou bronchiques méconnues iront en diminuant de nombre. Si les circonstances n'ont pas permis une réparation précoce, on peut espérer également que les médications actuelles permettront au patient d'échapper aux complications septiques qui le condamnent à peu près sûrement à l'exérèse pulmonaire.

EXPERIMENTAL SURGERY

EXPERIMENTAL STUDIES ON FIBROSIS OF THE SPHINCTER OF ODDI AND ON SPHINCTEROTOMY*

P. PRUDHOMME, M.D., P. NILOFF, M.D., F.R.C.S.[C], S. C. SKORYNA, M.D. and D. R. WEBSTER, M.D., F.R.C.S.[C], *Montreal, Que.*

THE EFFECTS of sphincterotomy on the physiology of the biliary tract and the pancreas have recently received considerable attention. Section of the sphincter of Oddi has been recommended mainly for the treatment of fibrosis of the sphincter and for the treatment of chronic recurring pancreatitis. Some surgeons, particularly Doubilet and Mulholland,[6] have reported success in treating pancreatitis by sphincterotomy, whereas others have not been able to obtain these results. Experimentally, Eiseman and his co-workers[10] demonstrated that the decrease of the common bile duct pressure after sphincterotomy is transitory and that the pressure soon returns to its preoperative level. They concluded that sphincterotomy could provide no lasting benefit in the treatment of pancreatitis. On the other hand, stenosis of the sphincter of Oddi due to fibrosis has been recognized as a cause of biliary tract symptoms, and some favourable results following sphincterotomy performed for this condition have been reported.

This study was undertaken to evaluate the effects of sphincterotomy on fibrosed sphincters. At the same time, we hoped that these studies might provide some information regarding the etiology of fibrosis of the sphincter of Oddi. In 1939, Branch, Bailey and Zollinger[4] demonstrated that extensive dilatation of the sphincter of Oddi in dogs produced fibrosis. Allen and Wallace,[1] and Colcock, Cattell and Pollack[5a, b] indicated that careful dilatation of the sphincter did not lead to fibrosis in humans. We desired to verify the effects of extensive dilatation experimentally and, if the procedure resulted in fibrosis, to perform sphincterotomies and study the results of this procedure. As far as we know, no reports have been published on the effects of experimental sphincterotomy on a previously fibrosed sphincter.

MATERIAL AND METHODS

In this study, 12 dogs, each weighing at least 50 lbs., were employed. The experiment was divided into three stages. In the first stage, a baseline biliary tract pressure with normal sphincters was obtained. In the second, the effects of dilating the sphincter were observed, and in the third, the effects of sphincterotomy were studied. Three methods of evaluation were employed: manometry, radiology and pathological examination. All operations were carried out under general anesthesia, using intravenous pentobarbital, and under sterile conditions.

In the first stage, the gallbladder was removed and choledochotomy with insertion of a short arm T-tube was performed. The gallbladder was removed to prevent errors in pressure readings. The T-tube was fastened to the abdominal wall by a nylon cannula of the type used by Eiseman *et al.*[10] and introduced through a stab wound. In the second stage, a duodenotomy was performed, and Ferris dilators up to the sizes of 5 and 7 mm. were introduced into the ampulla from below to stretch the sphincters extensively. This transduodenal approach was employed in order to avoid the removal of the T-tube from the common bile duct which could traumatize the common duct and disturb the baseline pressure readings. The third and final stage consisted of performing two types of sphincterotomy—some single-cut and some wedge resections. No sutures were employed in these sphincterotomies.

---

*From the Department of Experimental Surgery, McGill University, and the Department of Surgery, Jewish General Hospital, Montreal.

Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Quebec, January 1964.

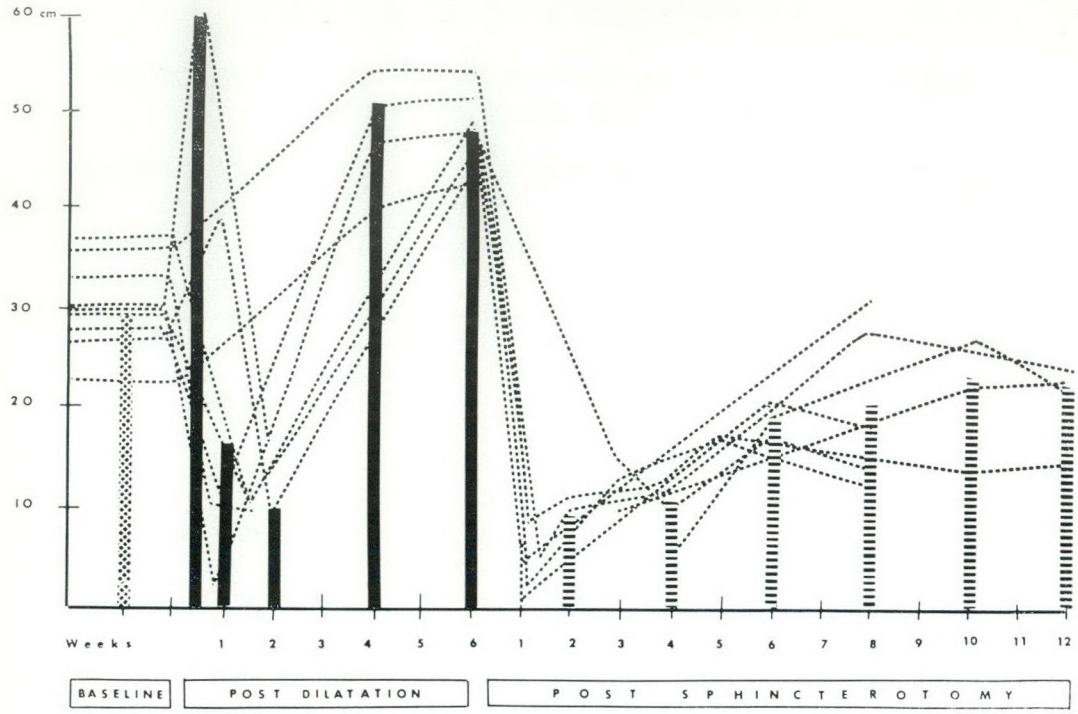


Fig. 1.—Composite graph showing baseline pressure readings compared with post-dilatation and post-sphincterotomy levels.

After each operation, pressure studies were recorded. The yielding pressure or sphincteric resistance is the pressure at which the sphincter will "give" under a perfusing system and allow the fluids to flow from the common bile duct into the duodenum. It is expressed in millimetres of water (or saline) as observed on an ordinary spinal manometer. Using a 30 c.c. syringe, saline was introduced into the T-tube at the rate of 20 c.c./min. and the gradual rise in pressure was observed on the water manometer. The point at which the pressure suddenly dropped owing to the yielding of the sphincter, allowing the passage of saline from the common bile duct into the duodenum, was interpreted as the yielding pressure. Ten such readings were taken each time and the average was recorded. All pressures were taken in fasting, unanesthetized dogs that were, as much as possible, free from surrounding distractions. The readings were taken at different intervals after all three surgical stages until a relatively constant level was obtained.

In attempting to correlate our pressure readings with anatomical changes in the biliary tree before and after the different stages of the experiment, T-tube cholangiograms were performed. In each case, 10 to 15 c.c. of a 60% solution of sodium and methylglucamine diatrizoate (Renografin 60) were instilled directly into the T-tubes; radiographs were taken and compared. All cholangiograms were performed under general anesthesia. Finally, at each operation and at autopsy, the area of the sphincter of Oddi was carefully observed and photographs were taken. Sections of the specimens were examined microscopically.

Results

Pressure readings after the first operation were relatively stable in any given dog after approximately three weeks (Fig. 1). The sphincter of Oddi in each dog was found to have a different resistance. The readings were in the vicinity of 260 to 350 mm. of saline. Immediately after stretching, no pressure readings could be obtained

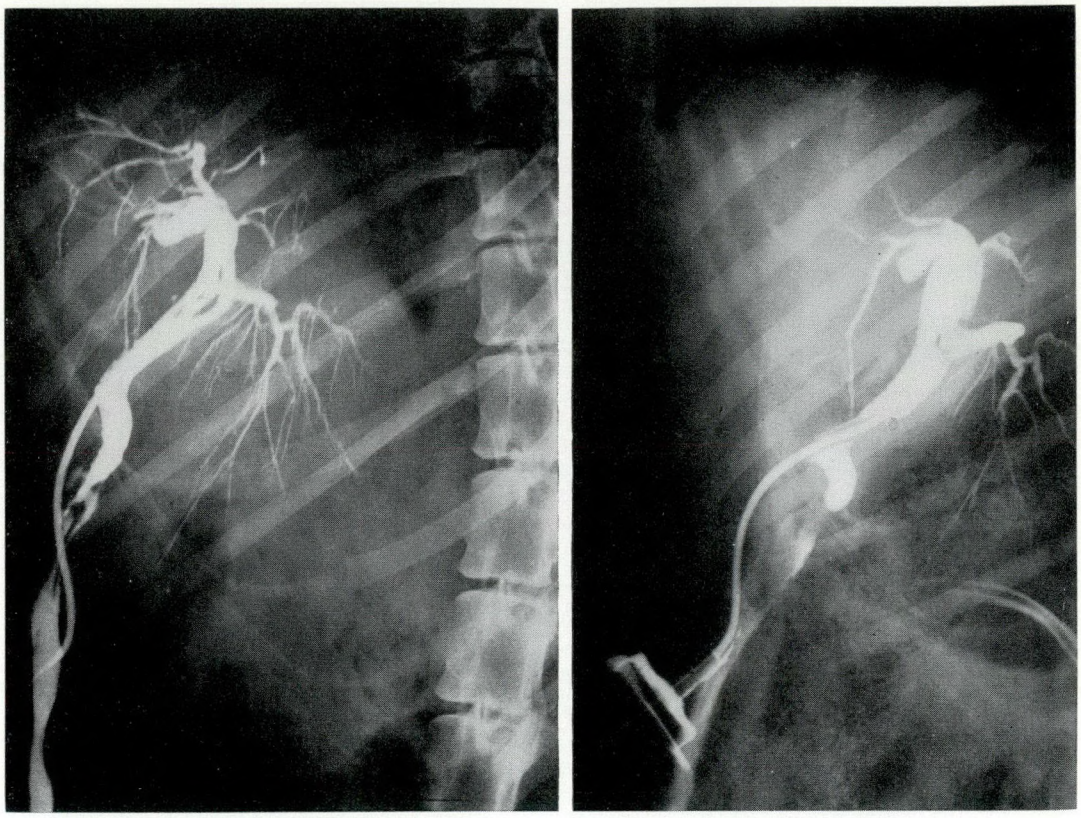


Fig. 2.—T-tube cholangiograms before and six weeks after dilatation.

because of the unopposed passage of bile into the duodenum. After a few days, the pressures rose to levels beyond the graduations on the water manometer. The rise was caused by marked inflammatory reaction and swelling of the papilla causing complete obstruction of the common bile duct. (This reaction was observed directly in a dog that died three days after the stretching of the sphincter.) After one week, the pressures were much lower than the predilatation readings; however, with time, they invariably rose above the original

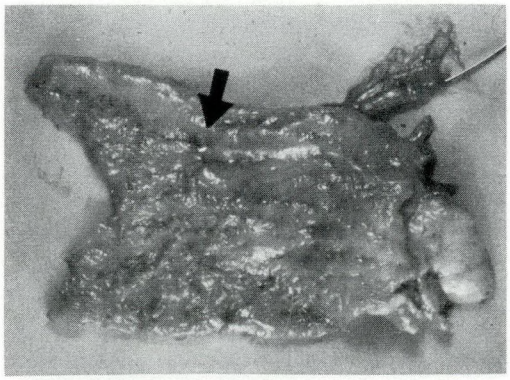


Fig. 3a.—Normal duodenal papilla. Note probe in orifice of ampulla of Vater.

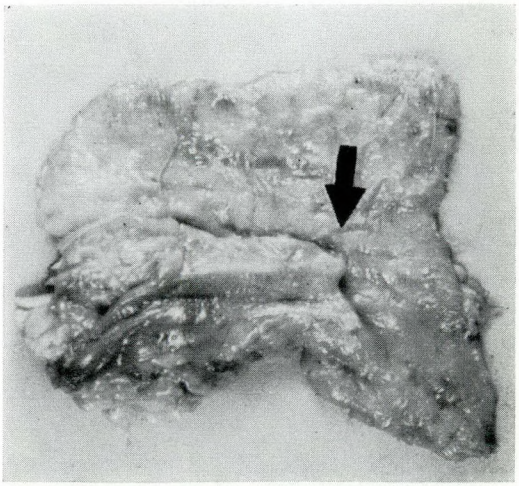


Fig. 3b.—Normal appearance of the choledochoduodenal junction (common bile duct opened longitudinally). Arrow points to choledochoduodenal junction.

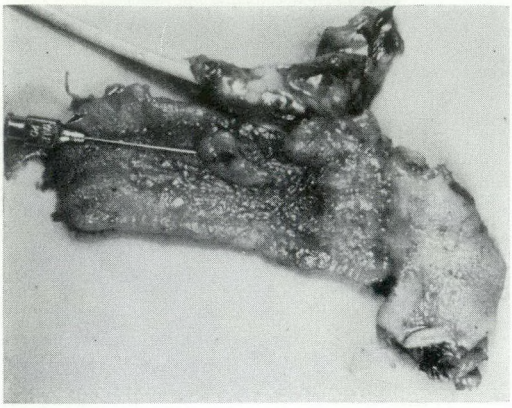


Fig. 3c.—Duodenal papilla six weeks after forced stretching. Note thickened scar to which needle points.

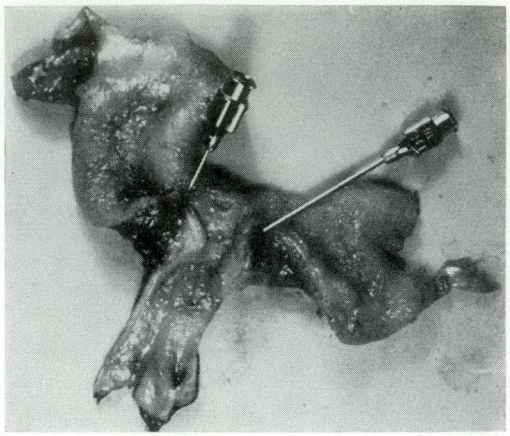


Fig. 3d.—Choledochoduodenal junction six weeks after forced stretching (common bile duct opened longitudinally). Note thickened scar to which needle points.

levels to as much as 200 mm. of water (Fig. 1).

Cholangiograms taken at this time and compared with those taken before stretching showed significant changes as well. The common bile ducts were found to be much wider, and the choledochoduodenal junctions were markedly narrowed. Passage of the contrast medium into the duodenum was decreased (Fig. 2). Four dogs were sacrificed at this stage and the areas of the sphincters showed marked thickening and induration (Fig. 3). Microscopically, fibrous tissue was prominent and muscle tissue almost non-existent (Fig. 4).

Finally, pressure readings were taken after sphincterotomy. After a few weeks, pressure readings were down again to lev-

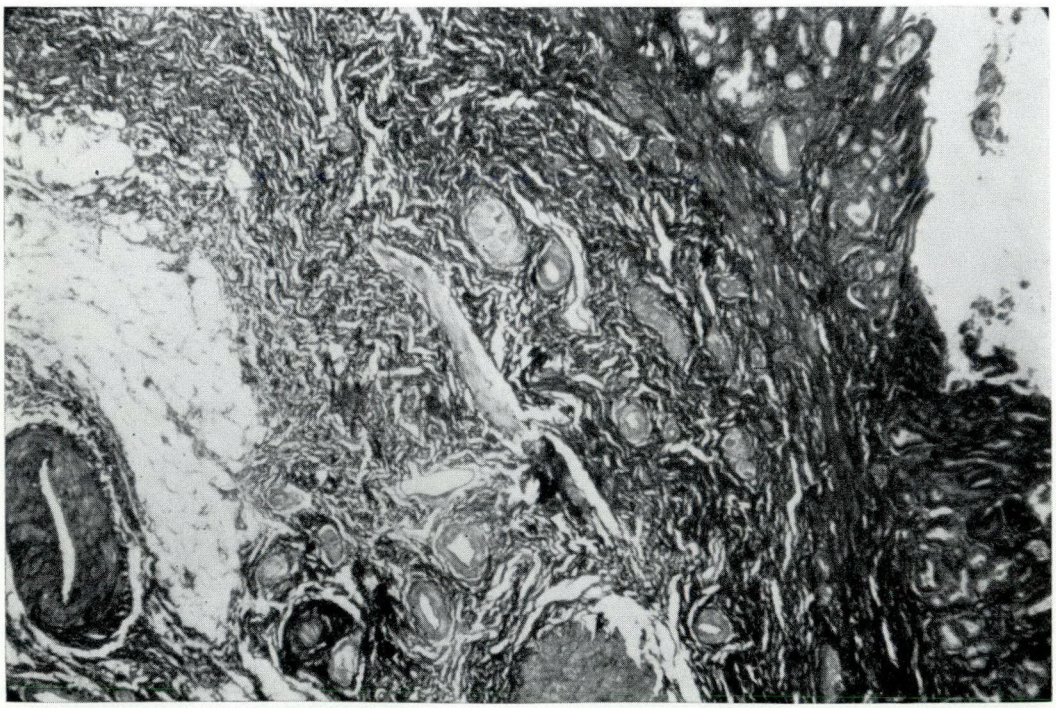


Fig. 4a.—Low-power microscopical appearance of the choledochoduodenal junction (trichrome stain to show fibrous connective tissue).

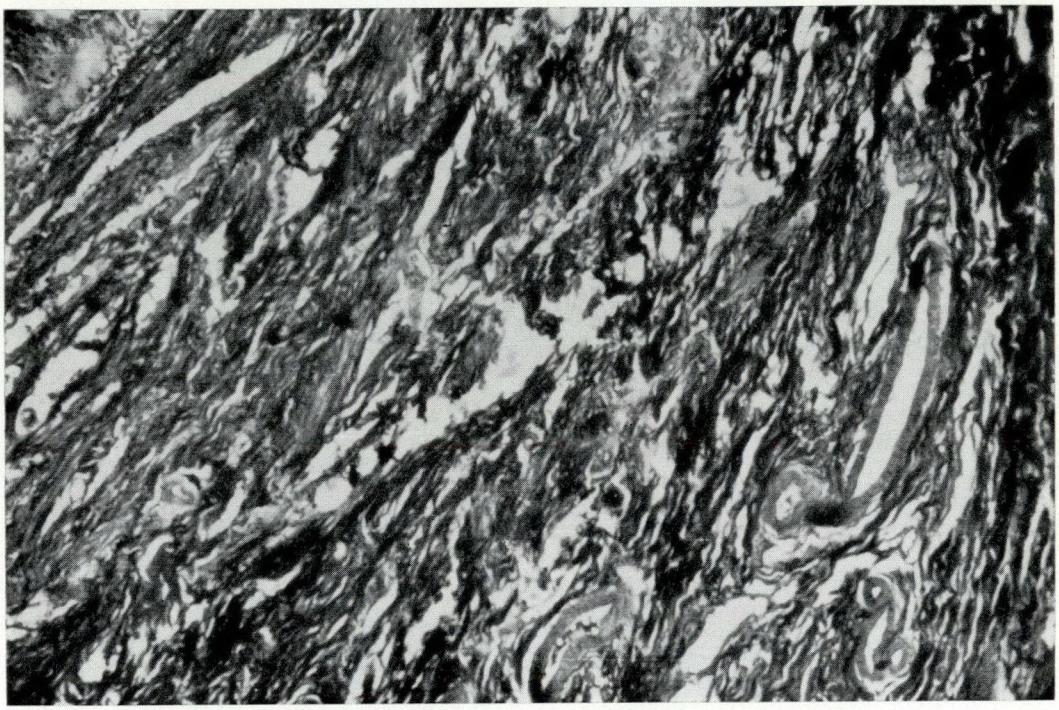


Fig. 4b.—High-power view of same area.

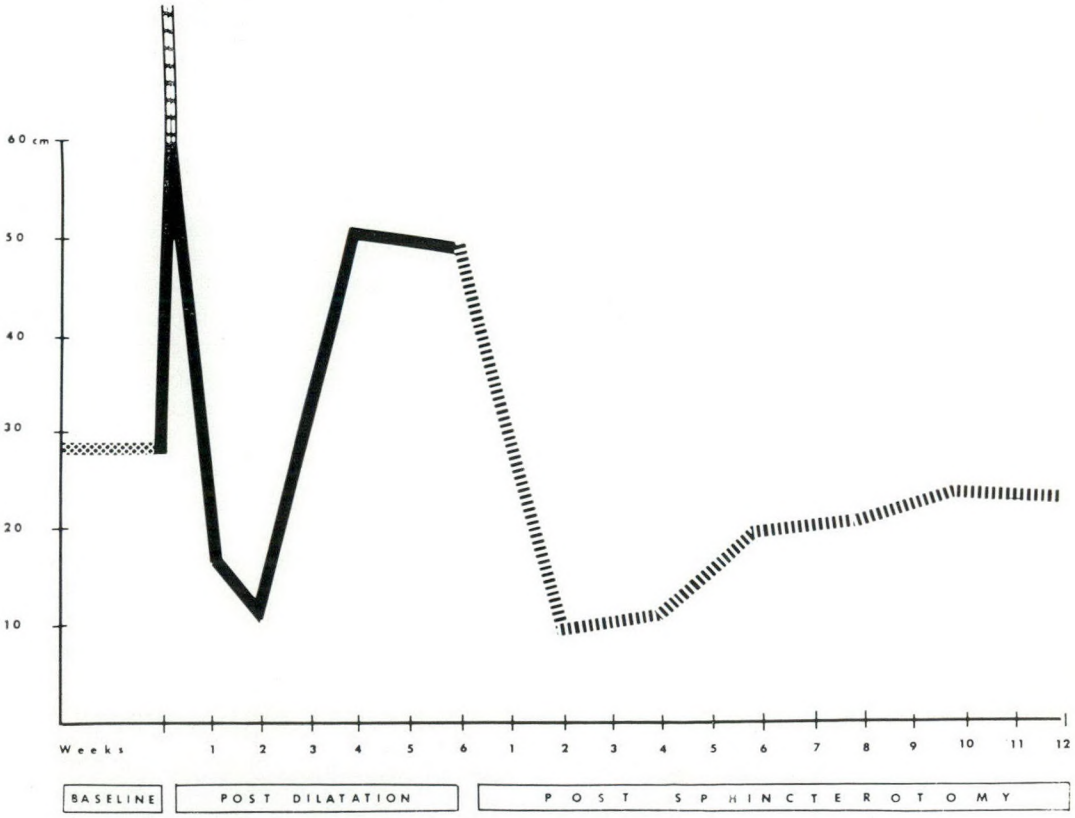


Fig. 5.—Composite graph of average manometric pressure readings.

Fig. 6.—Duodenal papilla 12 weeks after sphincterotomy showing patency and area of sphincterotomy.

els below the original ones, but during the weeks that followed they gradually increased and eventually became stable. These dogs were followed-up for 12 weeks after sphincterotomy. Although the final pressures were similar to the values obtained at the start of the experiment, in all cases they were much lower than those taken after forced dilatation with resulting fibrosis (Fig. 5). Cholangiograms were again performed and no evidence was obtained of obstruction to the passage of dye into the duodenum. At autopsy, the ampulla of Vater showed thickening and induration but the area of sphincterotomy could be clearly seen (Fig. 6), and large probes could be passed without difficulty.

Discussion

Oddi isolated a sphincter at the lower end of the common bile duct in 1887. The existence of this sphincter was confirmed by Boyden, who described its anatomy.[3, 9] Fibrosis of the sphincter is a definite pathological entity with clinical manifestations but its etiology is not well understood. Many factors such as chronic infection, repeated spasm and trauma from calculi or instrumentation have been mentioned as possible causes, but fibrosis has been recognized in patients without a history of such causes and, conversely, such conditions are often encountered without any evidence of fibrosis. Cattell and his co-workers[5] at the Lahey Clinic believe that stenosis of the sphincter of Oddi may exist as a primary entity in patients without any evidence of biliary or pancreatic disorders, and that it may be a cause of recurrent symptoms following cholecystectomy.

The first part of the present study demonstrates that forcible dilatation of the sphincter may produce fibrosis with resulting stenosis of the sphincter of Oddi and increase in intraductal pressure. Dilators of the size of the common bile duct may very well have this effect since the Vaterian system is normally much narrower than the common bile duct.[7, 9] All 12 dogs subjected to dilatation developed fibrosis as observed by an increase in yielding pressure in each case, increase in size of the common duct, and narrowing at the sphincter area as seen on radiographs and at autopsy.

The benefits derived from sphincterotomy and sphincteroplasty are also controversial. Eiseman's experimental findings supported by the clinical study of Thistlethwaite and Smith[16] are strikingly conclusive and suggest that the benefits of sphincterotomy are transitory. Nevertheless, good results following such procedures have also been reported.[2, 6, 8, 11-15] Eiseman's sphincterotomies were carried out on normal sphincters. All sphincterotomies in this study were performed upon fibrosed sphincters, where the muscular element was almost non-existent and was replaced

by fibrous tissue. Although the pressure readings slowly returned to levels similar to preoperative readings, in all cases they remained much lower than the post-dilatation levels. Sphincterotomy on the fibrosed sphincters was considered beneficial (Fig. 5).

Summary and Conclusions

The etiology of fibrosis of the sphincter of Oddi is not well understood and reports of the effects of sphincterotomy or sphincteroplasty are conflicting. This study was undertaken to verify the effects of extensive dilatation of the sphincter and to study the effects of sphincterotomy on fibrosed sphincters. For this purpose, 12 dogs were used and evaluation was made by means of manometry, radiology and pathological examination. After stretching, all sphincters became fibrosed. Sphincterotomy performed on eight dogs resulted in the lowering of yielding pressures in all cases. It is concluded that extensive and forced dilatation of the sphincter of Oddi results in the production of fibrosis and stenosis of the area. Furthermore, these findings support the conclusions of others that sphincterotomy performed on fibrosed sphincters has a beneficial effect.

References

1. Allen, A. W. and Wallace, R. H.: The surgical management of stone in the common bile duct, *Ann. Surg.*, **111:** 838, 1940.
2. Jones, S. A. and Smith, L. L.: Transduodenal sphincteroplasty for recurrent pancreatitis; preliminary report, *Ann. Surg.*, **136:** 937, 1952.
3. Boyden, E. A.: Anatomy of the choledochoduodenal junction in man, *Surg. Gynec. Obstet.*, **104:** 641, 1957.
4. Branch, C. D., Bailey, O. T. and Zollinger, R.: Consequences of instrumental dilation of the papilla of Vater; experimental study, *Arch. Surg. (Chicago)*, **38:** 358, 1939.
5. (a) Colcock, B. P.: Stenosis of the sphincter of Oddi, *Surg. Clin. N. Amer.*, **38:** 631, 1958.
5. (b) Cattell, R. B., Colcock, B. P. and Pollack, J. L.: Stenosis of the sphincter of Oddi, *New Eng. J. Med.*, **256:** 429, 1957.
6. Doubilet, H. and Mulholland, J. H.: Eight year study of pancreatitis and sphincterotomy, *J. A. M. A.*, **160:** 521, 1956.
7. Dowdy, G. S., Jr., Waldron, G. W. and Brown, W. G.: Surgical anatomy of the pancreatobiliary ductal system, *Arch. Surg. (Chicago)*, **84:** 229, 1962.
8. Edlund, Y. and Jacobsson, B.: Transduodenal sphincterotomy: analysis of operation cases, *Acta Chir. Scand.*, **113:** 476, 1957.
9. Eichorn, E. P., Jr. and Boyden, E. A.: The choledochoduodenal junction in the dog—restudy of Oddi's sphincter, *Amer. J. Anat.*, **97:** 431, 1955.
10. Eiseman, B. *et al.*: Sphincterotomy—an evaluation of its physiologic rationale, *A.M.A. Arch. Surg.*, **79:** 294, 1959.
11. Gillette, L.: External sphincterotomy for pancreatitis, *Ann. Surg.*, **138:** 24, 1953.
12. Major, J. W. and Ottenheimer, E. J.: Transduodenal sphincterotomy for chronic relapsing pancreatitis, *New Eng. J. Med.*, **248:** 130, 1953.
13. Nardi, G. L.: Technique of sphincteroplasty in recurrent pancreatitis, *Surg. Gynec. Obstet.*, **110:** 639, 1960.
14. Preston, D. J.: Transduodenal ampulla-duodenostomy for treatment of common bile duct obstructions without T-tube drain, *Surg. Gynec. Obstet.*, **100:** 499, 1955.
15. Riddell, D. H. and Kirtley, J. A., Jr.: Stenosis of the sphincter of Oddi; transduodenal sphincterotomy and some other surgical aspects, *Ann. Surg.*, **149:** 773, 1959.
16. Thistlethwaite, J. R. and Smith, D. F.: Evaluation of sphincterotomy for treatment of chronic recurrent pancreatitis, *Ann. Surg.*, **158:** 226, 1963.
17. Trommald, J. P. and Seabrook, D. B.: Benign fibrosis of the sphincter of Oddi: report of eight cases, *Western J. Surg.*, **58:** 89, 1950.

Résumé

L'étiologie de la fibrose du sphinctère d'Oddi est mal comprise, et les effets de la sphinctérotomie ou de la sphinctéroplastie donnent des résultats contradictoires. Le but de ce travail est de contrôler les effets de la dilatation forcée du sphinctère d'Oddi et, en même temps, d'étudier les effets de la sphinctérotomie sur les sphinctères fibrosés. A cette fin, 12 chiens ont été soumis à l'intervention et les résultats ont été étudiés par la manométrie, la radiologie, et l'examen pathologique.

Après dilatation forcée, les sphinctères sont devenus fibrosés. La sphinctérotomie, faite sur huit chiens a abouti, dans chaque cas, à un abaissement des pressions biliaires.

A la suite de ces résultats, il a été conclu que la dilatation forcée du sphinctère d'Oddi provoque une fibrose du sphinctère et, en plus, une sténose de la région. Ces observations confirment les effets avantageux de la sphinctérotomie dans les cas de fibrose du sphinctère.

STUDIES ON LAMBS OF THE DEVELOPMENT OF AN ARTIFICIAL PLACENTA

Review of Nine Long-Term Survivors of Extracorporeal Circulation Maintained in a Fluid Medium*

JOHN C. CALLAGHAN, M.D., B.Sc.(Med.), F.R.C.S.[C], F.A.C.S., M.S.,†
EARL A. MAYNES, M.D.‡ and HENRY R. HUG, M.D.,§ *Edmonton, Alta.*

THE possibility of supporting a new-born child in respiratory distress by means of a supply of oxygenated blood for extended periods of time led to the development of an artificial placenta as a means of testing such a system in an unanesthetized animal.[1, 2] In 1958, Westin, Nyberg and Enhörning[3] published their studies on seven previable human fetuses kept alive five to 12 hours by perfusion of the umbilical vein and hypothermia. In choosing the lamb as an experimental animal we were guided by the excellent studies of Dawes.[4] Much of our physiological information concerning fetal and new-born lambs was obtained from the works of Barcroft and Barron[5] and Barclay, Franklin and Prichard.[6] Harned *et al.*[7] suggested the use of a pump oxygenator in neo-natal respiratory distress syndrome, but did not publish details of their study in which they were able to maintain life in new-born lambs for up to an hour.

METHODS

This report is based on studies on nine lambs removed by Cesarean section from Suffolk ewes between 134 and 143 days of gestation (normal gestation period in this breed is 144 to 147 days) and placed in an artificial placenta. All of the animals reported in this communication are long-term survivors.

One ewe received intravenous pentobarbital (Nembutal) 1500 mg. as an anesthetic; the other eight received a spinal anesthetic consisting of 5 c.c. of 2% novocaine or lidocaine. With the ewe lying on the right side, a left muscle-splitting incision was made to provide access to the uterus. The hind legs of the fetus were identified by palpation and exteriorized as far as the groins through a small incision in the uterus. A polyethylene catheter was inserted into the femoral artery of the fetus for pressure and biochemical studies. The umbilical vessels were cannulated with inflow and outflow catheters. In each of the last five survivors, one femoral artery was used as the outflow vessel because its rate of flow proved to be more constant and was influenced less by vasospasm than were the umbilical vessels. As soon as the first cannula was inserted, heparin, 2 to 3 mg./kg. of estimated body weight, was given to the lamb.

In one experiment a rotating disc oxygenator was used; in five others a Medical-Monitors membrane oxygenator and Teflon membranes were used. In the last three experiments an oxygenator of our own design and silastic membranes were employed.

The artificial circulation was effected with the use of the equipment demonstrated in Fig. 1. The legend accompanying the figure identifies the various component parts. Two different extracorporeal circuits were used as depicted in Fig. 2.

With the Medical-Monitors oxygenator, the fetus was permitted to pump desaturated arterial blood into the oxygenator, situated above, and from this the blood was returned by gravity to the umbilical vein (Fig. 2b). Fig. 2a shows the circuit used for our last three experiments, whereby the fetus pumps arterial blood

---

*From the Surgical-Medical Research Institute, University of Alberta, Edmonton, Alta. Financed in part by Grant #608-7-35, Department of National Health and Welfare of Canada; The Department of Health, Province of Alberta, and the Alberta Heart Foundation.

Presented at the Annual Meeting of the Canadian Thoracic Society, Saint John, N.B., June 23-25, 1964.

†Chief of Thoracic and Cardiovascular Surgery, University of Alberta Hospital, Edmonton, Alta.

‡Former Fellow in Cardiovascular Research, Surgical-Medical Research Institute, University of Alberta.

§Fellow in Cardiovascular Research, Surgical-Medical Research Institute, University of Alberta.

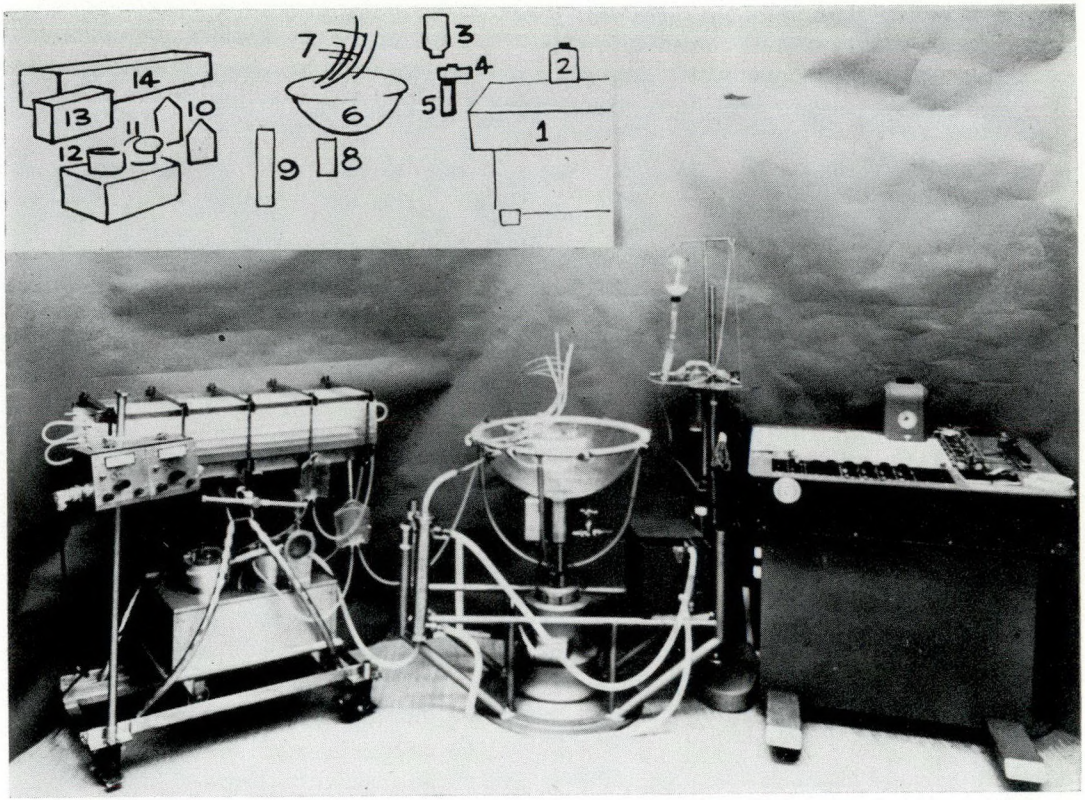


Fig. 1.—The experimental set-up. 1. Eight-channel recorder. 2. Registering time clock. 3. Strain gauge-saline. 4. Pressure strain gauges. 5. Mercury manometer. 6. Placental chamber. 7. Holding device with cannulas. 8. Artificial amniotic fluid temperature control. 9. Heat exchanger. 10. Venous and arterial reservoirs. 11. Blood filter. 12. Venous and arterial pumps. 13. Pump controls. 14. Membrane oxygenator.

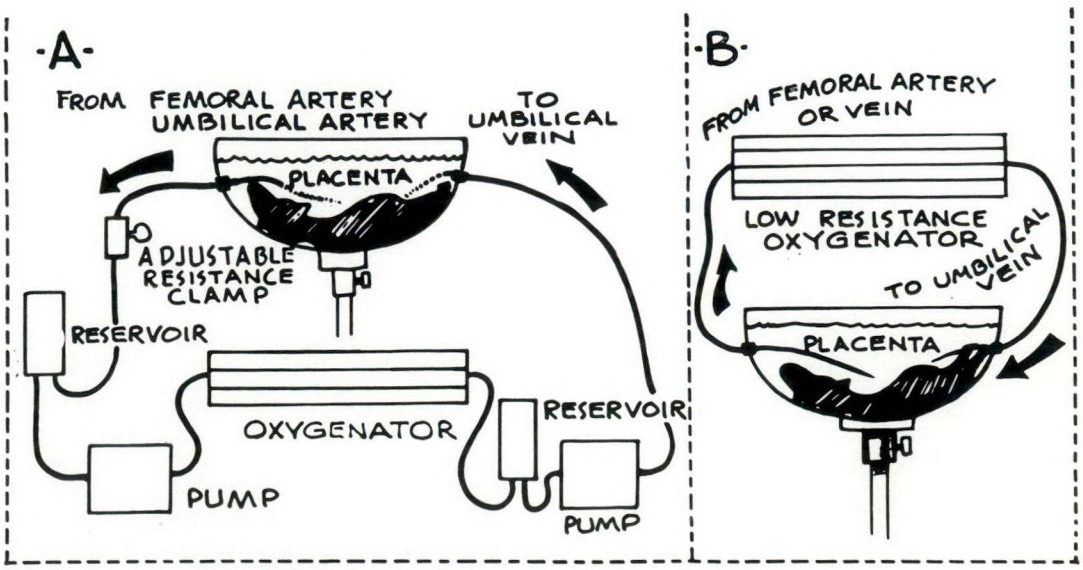


Fig. 2.—Two methods of artificial placenta circulation.

into a reservoir, the flow being controlled by partial occlusion with an excentric clamp. A Med-Science pump propels the blood through a rigid membrane oxygenator and into a second reservoir from which it is pumped into the umbilical vein.

After starting the perfusion, the umbilical cord was divided and the fetus was transferred to the placental chamber, care being taken to avoid exposing the head of the fetus to the atmosphere. The animal was then submerged in artificial amniotic fluid containing glucose, Cl^-, Na^+ and K^+ ions in concentrations similar to those found in the amniotic fluid of sheep.

Pressures in the femoral artery were continuously measured by Statham pressure transducers* and recorded on an eight-channel macropolygraph.† Oxygen saturation in the blood was measured by the Thomas-Van Slyke method in our early experiments and later with an A-O Oximeter, Model 10800.‡ The pO_2, pCO_2 and pH were studied with the Epsco-Medico Blood Parameter Analyzer.§ Hemoglobin, plasma hemoglobin, blood urea and blood sugar levels were estimated on a Klet colorimeter.¶ The Na and K measurements were made by flame-photometric methods, and chloride determinations by the Schales and Schales method.

Results

The weights of the nine lambs used in this study varied between 2000 and 5100 g. Perfusion times ranged from 23 to 165 minutes before the animal was delivered into the atmosphere (Fig. 3).

In the early experiments when the umbilical arteries were used as the outflow source, the time for insertion of catheters was critical, since during these maneuvers the blood supply which the fetus obtains from the placenta is greatly reduced.[8] This problem has been remedied in our last five successful perfusions by using one femoral

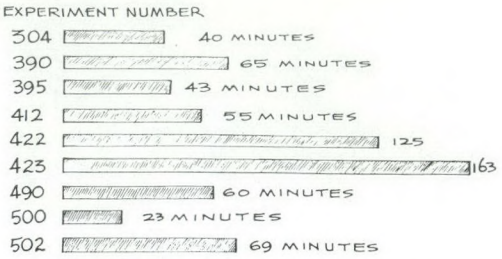


Fig. 3.—Duration of nine long-term survivors in the artificial placenta.

artery, which can be cannulated without disturbing the lamb's own placental blood supply. In all cases the return blood flow was through the umbilical vein.

The arterial outflow rates as measured in seven animals are shown in Fig. 4a. The flow rates varied between 13 and 100 c.c./kg. per minute. The pulse rates during perfusion varied between 80 and 250 per minute (Fig. 4b). Other experimental animals that had periods of bradycardia below these levels failed to survive.

The oxygen tension in the umbilical artery was measured in only four animals and is shown in Fig. 4c. The umbilical artery pO_2 remained below 50 mm. Hg in all animals. The umbilical vein pO_2 was kept at high levels so as to fully saturate the blood and compensate for a lower-than-normal flow in supplying the animal's oxygen requirements.

The pCO_2 values are depicted in Fig. 4d; these show a wide range of values. The range in pH is shown in Fig. 4e.

The systolic femoral artery pressure varied between 30 and 135 mm. Hg (Fig. 4f). As in the case of the pulse rate, animals experiencing periods of hypotension below these levels did not survive. After delivering the animals to the atmosphere, the blood pressure rose by 10 to 30 mm. Hg above that recorded during the last few minutes of placental circulation.

Blood glucose levels were kept between 50 and 150 mg. % by the addition of appropriate amounts of 50% glucose solution at regular intervals. Preperfusion and postperfusion determinations of Cl^-, Na^+ and K^+ were done in two experiments and the values remained unchanged.

*Statham Instruments Inc. of Puerto Rico, Hato Rey, Puerto Rico.

†Gilson Medical Electronics, Middleton, Wisconsin.

‡American Optical Company, Buffalo, New York.

§Epsco, Inc., Cambridge, Mass.

¶Van Waters & Rogers of Canada Ltd., Vancouver, B.C.

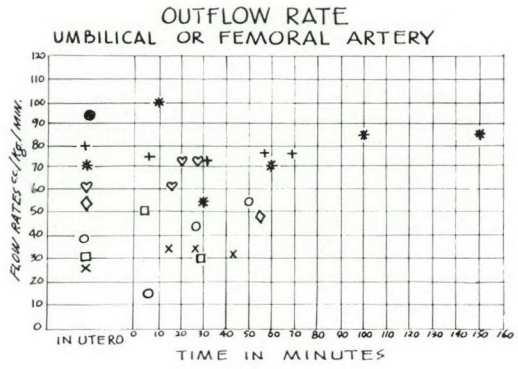


Fig. 4a

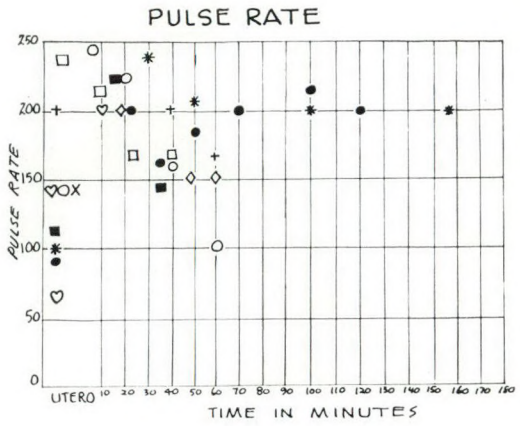


Fig. 4b

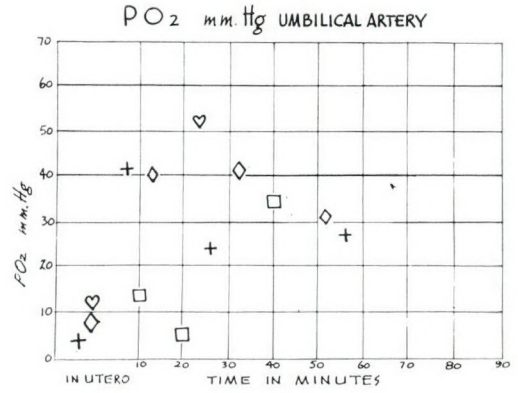


Fig. 4c

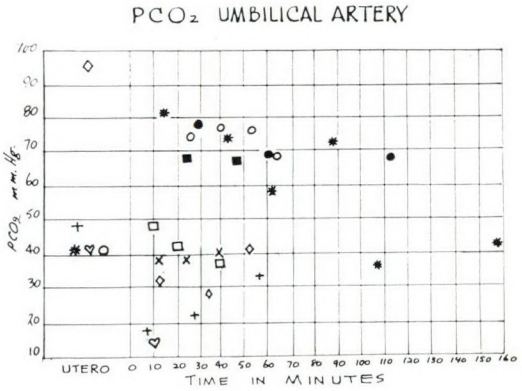


Fig. 4d

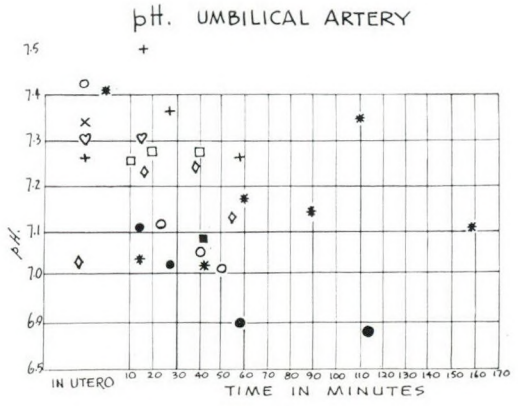


Fig. 4e

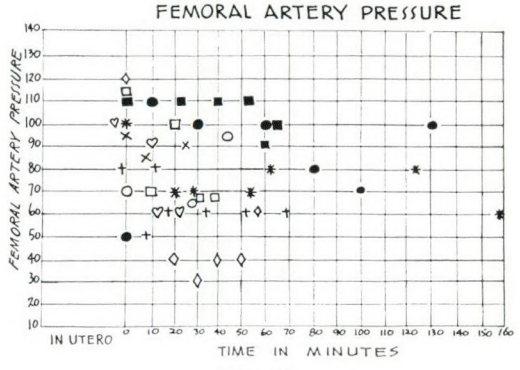


Fig. 4f

Figs. 4.—a-f

Each of the symbols used in Figs. 4a-f represents one of the nine experimental animals.

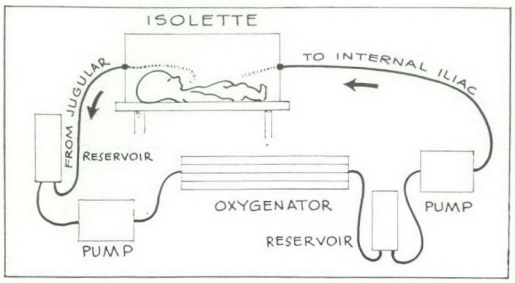


Fig. 5.—Circuitry for cardiopulmonary support of postnatal circulation.

Discussion

In our early studies,[1, 8] we were able to keep lambs alive in the artificial placenta for periods of up to 19 hours. These animals, however, failed to survive for more than a few hours after exposure to air.

There are undoubtedly many reasons why the animals deteriorated after even short periods in the placenta. Prolonged time for cannulation was a common cause for failure in the experience of Harned *et al.*[7] as well as in ours.[8] Cannulation of a femoral artery has obviated this difficulty.

The flow rates that we observed during perfusion varied between 13 and 100 ml./min./kg. Adams and Lind[9] found that flow rates in the human fetus changed from 60 ml./kg./min. just before birth to 273 ml./kg./min. just after birth. Dawes[4] in his study demonstrated umbilical flows of 100 to 180 ml./kg./min. as an average in six mature lambs. He designed a study in which an artificial lung was placed in the circuit with a heart-lung preparation of a lamb, and demonstrated a decrease in size of the ductus arteriosus if arterial oxygen saturation rose as high as 76%. In none of our animals did the peripheral arterial tension rise above 50 mm. Hg.

The oxygen saturation values of the blood returning to the umbilical vein were kept at or near 100%, rather than at the normal value of 85% defined by Dawes, in order to compensate for a reduction in flow.

In investigating the possible clinical application of this procedure we have perfused dogs by a veno-venous type of circulation, with survival, for periods of up to two and a half hours.[10, 11] More recently by means of a veno-arterial type of bypass (Fig. 5) that requires the cannulation of only one jugular vein and internal iliac artery, we have been able to keep new-born lambs alive for periods of up to five hours while in an isolette breathing less than 2% oxygen.

This latter technique is proving to be more practical for the postnatal circulation. Our preliminary results indicate that veno-arterial perfusion from a membrane oxygenator may possibly provide a practical method of support for infants suffering from respiratory distress syndrome of the newborn.

Summary

The use of extracorporeal circulation and oxygenation of blood as a form of treatment for respiratory distress of the newborn is considered feasible.

In order to study such a system, an artificial placenta was developed and unborn lambs were used as experimental animals. Nine surviving animals were studied in detail during perfusions ranging in duration from 23 to 165 minutes. Pulse, blood pressure and flow rates were continuously monitored so that the optimum levels of these parameters could be determined. Also, pH, pO_2 and pCO_2 measurements were made at frequent intervals.

The use of extracorporeal circulation requiring the cannulation of only one jugular vein and internal iliac artery in a system that eliminates the blood-gas interface by means of a membrane oxygenator shows promise in the clinical field.

We are grateful to Messrs. J. Fortin, P. Tretiak, C. Dryden, R. Grant and R. Blackburn for their enthusiastic technical assistance, to Miss S. Kerpan for preparation of the manuscript, to Mr. Meredith Evans for the art work, and to our shepherd, Mr. R. Richmond.

References

1. Callaghan, J. C. and Angeles, J. D.: Long-term extracorporeal circulation in the development of an artificial placenta for respiratory distress of the newborn, *Surg. Forum*, **12:** 215, 1961.
2. Cardozo, D. W. and Callaghan, J. C.: An approach to cardiovascular and respiratory distress in infancy, *Alberta Med. Bull.*, **25:** 11, 1960.
3. Westin, B., Nyberg, R. and Enhörning, G.: Technique for perfusion of the previable human fetus, *Acta Paediat. (Uppsala)*, **47:** 339, 1958.

4. Dawes, G. S.: Changes in the circulation at birth, *Brit. Med. Bull.*, **17:** 148, 1961.
5. Barcroft, J. and Barron, D. H.: Blood pressure and pulse rate in the foetal sheep, *J. Exp. Biol.*, **22:** 63, 1945.
6. Barclay, A. E., Franklin, K. J. and Prichard, M. M. L.: The foetal circulation and cardiovascular system, and the changes that they undergo at birth, Blackwell Scientific Publications, Oxford, 1944.
7. Harned, H. S., Jr. *et al.*: Use of the pump oxygenator to sustain life during neonatal asphyxia of lambs, *A.M.A. Amer. J. Dis. Child.*, **94:** 530, 1957.
8. Callaghan, J. C. *et al.*: Studies in the development of an artificial placenta: the possible use of long term extracorporeal circulation for respiratory distress of the newborn, *Circulation*, **27:** 686, 1963.
9. Adams, F. H. and Lind, J.: Physiologic studies on the cardiovascular status of normal newborn infants (with special reference to the ductus arteriosus), *Pediatrics*, **19:** 431, 1957.
10. Callaghan, J. C. *et al*: Study of prepulmonary bypass in development of an artificial placenta for prematurity and respiratory distress syndrome of the newborn, *J. Thorac. Cardiov. Surg.*, **44:** 600, 1962.
11. Callaghan, J. C., Cardozo, D. and Boracchia, B.: Study of prepulmonary bypass to replace the function of the lungs for long periods in experimental animals, *In*: World Congress of Cardiology, 4th, Mexico City, Mexico, 1963.

Résumé

La possibilité de traiter les nouveau-nés asphyxiques par l'administration de sang oxygéné a conduit au développement du placenta artificiel. Cet article résume les résultats expérimentaux des auteurs, qui ont travaillé sur des fétus de mouton accouchés par césarienne et placés sous contrôle du placenta artificiel. Neuf de ces animaux, pesant entre 2000 et 5100 g. survécurent très longtemps. La circulation artificielle fut établie grâce à des cathéters de polyéthylène insérés dans l'artère fémorale et les vaisseaux ombilicaux; la coagulation était prévenue par l'administration d'héparine au fétus. Les oxygénateurs utilisés furent du type à disque rotatif ou bien du type à membrane. La durée de la perfusion varia entre 23 et 165 minutes, et son débit entre 13 et 100 ml. par minute par kg. Divers examens de laboratoire purent être pratiqués, parmi lesquels: pH sanguin, pCO_2, pO_2 et glucose. Il ressort de ces expériences que le facteur important dans le succès de cette réanimation est la vitesse de la canulation des vaisseaux. Récemment, les auteurs ont procédé à des expériences similaires chez le chien en utilisant la veine jugulaire et l'artère iliaque interne. Ces derniers résultats suggèrent d'intéressantes possibilités d'application à la clinique humaine.

TEXTBOOK OF SURGERY. Edited by David A. Macfarlane and Lewis P. Thomas. 759 pp. Illust. E. & S. Livingstone Ltd., Edinburgh; The Macmillan Company of Canada Limited, Toronto, 1964. $10.75.

A new textbook of surgery should be an exciting event because it implies that existing books have in some way failed to meet a need, and the new book will be better either in a conventional way or will have a new approach. The present textbook, the authors state, is primarily directed at students, to provide them with concise and didactic surgical information, in logical sequence, as an introduction for further study. To make the price reasonable, line drawings only have been provided, and rather than tabulate, an attempt has been made to make the book easy to read, with conformity of style of the contributors. Thus, the present book is in the conventional class, and it remains to be seen if the authors have been successful in making it better.

In fact, the book is a good one in its class; the information in the text is sound and it is easy to read, but it is sad that on reading this book one is left with the impression that in surgery all problems are solved, treatment is definitive and failures are few. In vain one looks for a hint that surgeons differ, or an encouragement to an enquiring mind for further study. This feeling is reinforced by the references given at the end of each chapter. Only two or three are given, and of all, only 16 are to original articles, the remainder being to other textbooks or monographs. The world literature is ignored to the extent that only nine of the references come from sources other than Great Britain.

The illustrations are unsatisfactory. They are too diagrammatic; they contain errors, such as ACTH shown being secreted by the adrenal in Fig. 50, or are inadequate, as in Fig. 39 relating to breast lymphatics. They are not suitable substitutes for good reproductions of radiographs. North American readers may have some difficulty with the trade names of British drugs, when the generic name is not given. It is a pity to see crystalline penicillin G excluded from the list of common chemotherapeutic agents.

Although the authors have succeeded in their aim, it is doubtful whether this textbook would appeal to the more thoughtful student, to North American readers because of the inadequacies of the references and names of drugs, or to the practising doctor because of its limitations. It would probably be of value in acquiring enough knowledge to pass a qualifying exam.

FAT EMBOLISM: ITS PRODUCTION AND SOURCE OF FAT*

K. S. MORTON, M.D., C.M., F.R.C.S.[C] and M. J. KENDALL, M.Sc., *Vancouver, B.C.*

According to Groskloss,[6] fat embolism in man was first described by Muller in 1860, and since that time a great deal has been written about this intriguing clinical entity. Although more recent investigations into this subject have been transferred to the laboratory, the majority of reports are still based on clinical and pathological studies. We have been able to produce pulmonary fat embolism in rats and rabbits by fracture and by soft-tissue injury, and in addition have reached certain conclusions regarding the source of fat in such embolization.

I. Production of Pulmonary Fat Embolism in the Rat

In the literature on experimental fat embolism, few workers have used fracture as the initiating agent for embolism and to our knowledge none has used the rat as the experimental subject. Peltier[14, 15] fractured femurs of rabbits and tibias of dogs and found fat emboli in blood and in lungs, kidneys and brain. The degree of fat embolism was, however, not described. Johnson and Svanborg,[7] using rabbits, found fat embolism as commonly in soft-tissue injury as in fracture, but again did not describe the degree of fat embolism produced. Successful techniques using fracture have also been reported by Kane *et al.*[8] (rabbit), Downing[3] (goat) and Swank and Dugger[16] (rabbit). It remained for Glas, Musselman and Davis,[5] however, to state precisely the degree of pulmonary fat embolism to be expected by femoral fracture in the rabbit.

In the experiments reported in this communication, the criteria for the production of fat embolism were based on histological examination of frozen sections of lung, which were 35 microns thick and were stained with Sudan IV. All emboli appearing within alveolar walls (but not alveolar spaces) in a full cross-section of one lung were counted. Pulmonary embolism was graded by a modification of the method of Glas *et al.*[5]: Grade 0, no emboli; Grade 1, 1 to 4 emboli; Grade 2, 5 to 10 emboli; Grade 3, 11 to 49 emboli; Grade 4, 50 to 100 emboli; Grade 5, loaded.

It might be stated at the outset that renal glomeruli were also examined for fat emboli but in no instance were such emboli detected in animals subjected to various types of trauma.

Experimental Results

TABLE I.—Fat Embolism in Closed Fracture

	Test animals (20)	*Control animals (6)*
Average grade	2.3	0.3
Number		
Range	0-49 emboli	0-2 emboli
Average	8.8 emboli	0.5 emboli

The grade stated represents an average figure determined from a range of 0 to 3 in test animals, 0 to 1 in control animals.

1. Closed fracture—26 rats.—In 20 animals, closed fractures of both femurs and both tibias were produced manually under pentobarbital (Nembutal) anesthesia. Six control animals were anesthetized with pentobarbital but suffered no fracture. Twenty-four hours later the animals were killed and the lungs were examined histologically. The degree of pulmonary fat embolism produced is shown in Table I.

2. Closed fracture and heat—56 rats.—In an effort to increase the degree of pulmonary fat embolism obtainable by closed fracture, animals were held in an environment of 100° F. for one-half hour before and one-half hour after the closed fracture was produced. There were 36 test animals (some with two fractures, others with four) and 20 controls. All were sacrificed at 24 hours. The results of these procedures are shown in Table II. Although average figures were the same in animals with two and with four fractures, the highest de-

*From the Trauma Research Unit, Department of Surgery, University of British Columbia, Vancouver, B.C.

This work was supported by Defence Research Board Grant No. 66-9304 and was based on material presented to the Regional Meeting of the Royal College of Physicians and Surgeons of Canada, Vancouver, B.C., October 23 and 24, 1964.

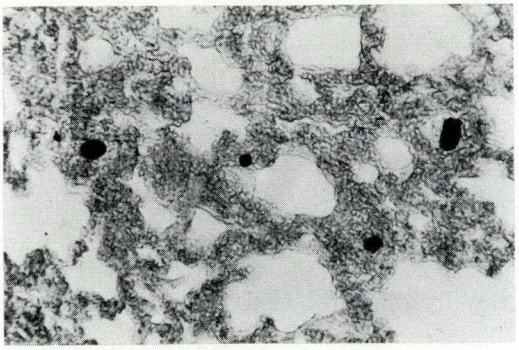


Fig. 1.—Maximum pulmonary fat embolism in closed fracture and heat (Sudan IV stain).

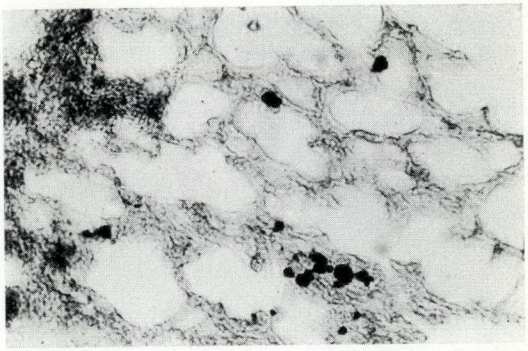


Fig. 2.—Open fracture and heat. Lungs loaded with fat emboli (Sudan IV stain).

gree of pulmonary fat embolism occurred in an animal with four fractures (Fig. 1). The difference between control and test figures shows a high degree of significance when subjected to statistical analysis (>0.005).

TABLE II.—Fat Embolism in Closed Fracture and Heat

	Test animals		
	Two fractures (10)	*Four fractures (26)*	*Control animals (20)*
Average grade	2.7	2.7	0.7
Number			
Range	0-100 emboli	0-204 emboli	0-14 emboli
Average	23 emboli	30 emboli	1.8 emboli

3. Closed fracture, heat and chlorpromazine—11 rats.—On the basis of its vasodilator action,[9] this drug was administered to a small series of rats subjected to fracture and heat. No increase in the degree of pulmonary fat embolism was obtained.

4. Open fracture and heat—37 rats.—An attempt to produce a more consistent and greater degree of pulmonary fat embolism was made by controlling the type of fracture produced and limiting damage to vascular channels. Accordingly, fractures were produced by making an incision over the lower femur and upper tibia and inserting fine-nosed pliers to produce comminuted fractures. Animals were again held in an environment of 100° F. for one hour and sacrificed at 24 hours.

In five test animals, lungs were loaded with fat emboli (Fig. 2) and in the remaining 25 test animals the average grade of pulmonary fat embolism was 4, that is, an average of 78 emboli per lung section was produced (range 5 to 149) (Table III).

TABLE III.—Fat Embolism in Open Fracture and Heat

	Test animals (25)	*Control animals (7)*
Average grade	4	1
Number		
Range	5-145 emboli	1-3 emboli
Average	77.8 emboli	1.3 emboli

Control animals exposed only to heat. Grade represents an average figure with a range of 2 to 5 in test animals.

In spite of this wide range, these figures are again highly significant ($> .01$).

5. Soft-tissue injury—82 rats.—In 31 animals, soft-tissue injury was produced by binding the hind limbs with heavy string (Fig. 3) for a period of three to four hours. Observations of pulmonary emboli were made 24 hours later and are recorded in Table IV.

TABLE IV.—Fat Embolism in Soft-Tissue Injury

	Test animals		
	Soft-tissue injury (31)	*Soft-tissue injury and heat (28)*	*Control animals (heat) (23)*
Average grade	2.7	3.0	1.0
Number			
Range	3-42 emboli	10-102 emboli	0-8 emboli
Average	18 emboli	28.5 emboli	2.8 emboli

In a further series of 28 rats subjected to both a warm environment and soft-tissue injury to hind limbs, pulmonary fat embolism was much increased (Table IV) and in both series greatly exceeded the findings in 23 control animals.

Statistical analysis of the differences between the heat controls and the soft-tissue injury group reveals a high degree of sig-

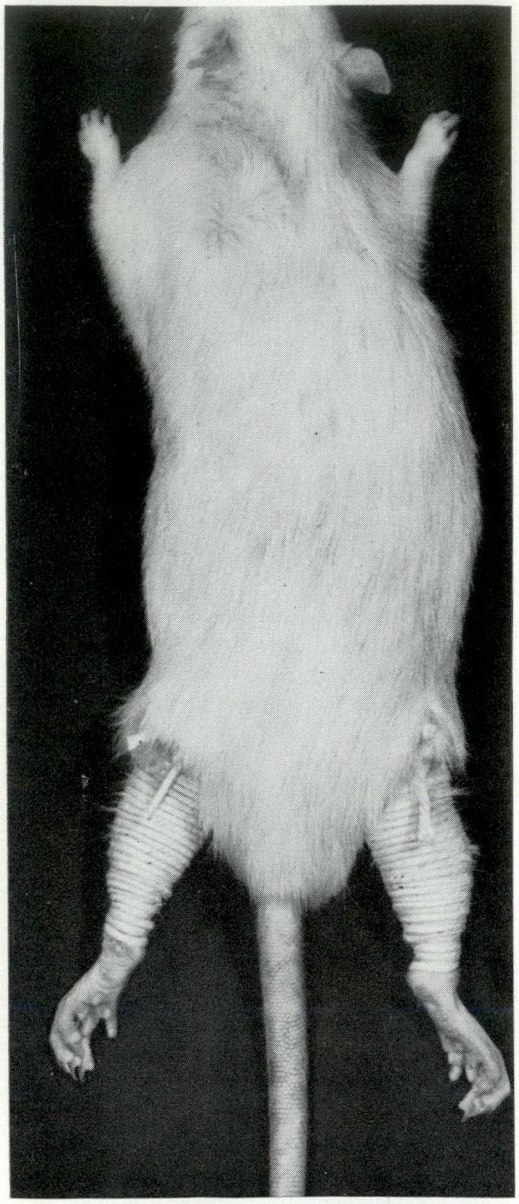


Fig. 3.—Method of producing soft-tissue injury by binding hind limbs with string.

nificance (>0.01), and between heated soft-tissue injury and heat controls a very high degree of significance (>0.0005), but no significant difference between soft-tissue injury and heated soft-tissue injury.

Discussion

Although it is clear that simple closed fracture of the four major hind-limb bones of rats produces a degree of pulmonary fat embolism, the results so obtained are not consistent and the degree of embolism is not marked. Both of these factors, consistency and degree, are increased to a significant degree by placing animals with fractures in a warm environment, but the mechanism of enhancement is not clear. Heat was used to produce vasodilatation but it is possible that it reduced the viscosity of the body fats allowing them freer entry into open veins. That heating aggravates hypotensive shock and lowers venous pressure (both of which are assumed to accompany the severe fractures) is discounted by our observations of normal blood pressures in a small series of animals similarly treated and by our consistent failure to produce pulmonary fat embolism by inducing hypotension.[13]

With regard to vasodilatation, it is of interest that chlorpromazine failed to enhance the degree of pulmonary fat embolism produced by closed fracture. It is therefore suggested that the increased environmental temperature does not enhance pulmonary fat embolism either by vasodilatation or by aggravation of hypotension.

By virtue of its less-refined technique, closed fracture tends to produce greater damage to venous channels draining the fracture site. The degree of embolism so produced is thereby limited. Open fractures performed under direct vision and therefore less likely to damage the larger venous channels, produce by far the greatest degree of fat embolism particularly when this procedure is performed in a warm environment. Statistical analysis indicates that this difference is significant (>0.01).

It is tempting to conclude that, with lowered viscosity of body fats in a warm environment and selective comminuted fractures of four major bones sparing damage to large venous channels, pulmonary fat embolism is given its best chance to occur. However, the fact that soft-tissue injury alone produces fat embolism greatly weakens this conclusion. Although no examination for patency of veins in these crushed limbs could be carried out, it seems certain that complete, tight binding of the limbs for three to four hours would indeed damage the endothelium of veins,

in many instances leading to permanent collapse or thrombosis.

The fact that soft-tissue injury without fracture produces pulmonary fat embolism, although to a slightly less degree than with fracture, confirms the reports of Swank and Dugger[16] and Johnson and Svanborg.[7] Although damage to fat depots in these crushed limbs is apparent, one can less easily postulate torn and open veins that would allow entry to fat freed from within fat cells. Thus the clinical appearance of the syndrome is again closely paralleled in the laboratory animal.

II. The Source of Fat in Fat Embolism

Gauss,[4] in 1924, suggested the mechanical theory of pathogenesis, that is, that fat liberated from the marrow by fracture enters ruptured veins held open by rigid bone and affects first the lungs and then the systemic circulation to produce the classical signs and symptoms. The first criticism of this concept was that of Lehman[10] in 1929, who suggested the biochemical theory: that is, the physiological emulsion of fat in plasma may, through physicochemical changes in blood, become so altered that agglomeration of ultramicroscopic particles occurs to a degree which allows fat droplets to plug capillaries. The controversy continues between the proponents of the two theories, supported on one side by the work of Peltier[15] and on the other by that of Johnson and Svanborg,[7] LeQuire *et al.*,[11] Bergentz[2] and Adkins and Foster.[1] The study reported in this section of the paper was designed to test the validity of the mechanical theory in two experiments in 28 rats and 25 rabbits.

Again, the criterion for the production of fat embolism was histological examination of frozen sections of lung 35 microns thick, stained with Sudan IV and graded as before. Although kidneys were routinely examined in the same way, fat emboli were in no instance observed in renal glomeruli after fracture.

Experimental Results

1. *Fracture and tourniquet—28 rats.*— Sixteen control animals each with four open fractures produced by the technique described above were held in a warm environment for one hour and sacrificed six hours later. Each developed an average of 64.2 emboli per lung section, or Grade 4 embolism (Table V). In 12 test animals, tourniquets were placed above the fractures in an effort to block the venous drainage effectively. These tourniquets consisted of a series of through-and-through massive ligatures of the soft tissues of the upper thigh (Fig. 4), which were found after trial of many techniques to be the only method which avoided slipping of the tourniquet and allowed inclusion of all possible sources of venous return from the damaged limb. Tourniquets were left in place for the full duration of the experiment (six hours). Lungs examined for fat emboli at this time revealed an average of 10.4 emboli per lung section, or an average of Grade 2 embolism (Table V). The difference between this figure and that for the control group is highly significant, when subjected to statistical analysis (>0.0005).

TABLE V.—Fat Embolism in Fracture with Tourniquet

	Test animals (fracture and tourniquet) (12)	*Control animals (fracture) (16)*
Average grade	1.9	3.6
Number		
Range	1-51 emboli	7-160 emboli
Average	10.4 emboli	64.2 emboli

Average grade represents a range of 1 to 4 in test animals, 2 to 5 in control animals.

2. *Examination of venous blood from fractured and intact limbs—25 rabbits.*— The source of intravascular fat emboli was studied in 25 rabbits in which venous blood from a hind limb (of which femur and tibia were fractured) was examined and compared with blood from the contralateral intact hind limb. In anesthetized rabbits with retrograde polyethylene catheters placed in the right and left femoral veins, blood samples were drawn before and at appropriate intervals after fracture of the femur and tibia on one side and from the contralateral intact limb. Fractures were open and were performed under direct vision through a small incision to minimize

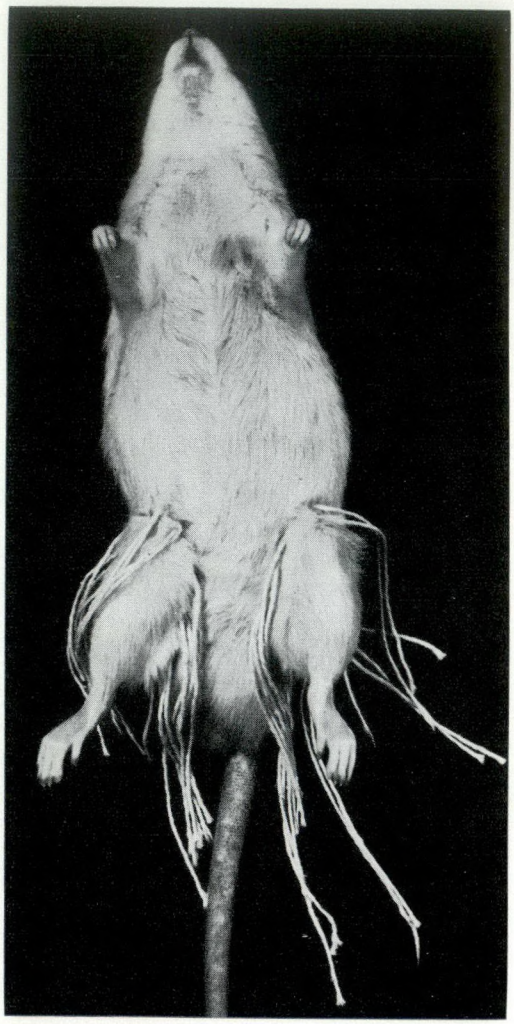


Fig. 4.—Tourniquets consisted of multiple through-and-through ligatures proximal to fractures.

trauma to soft tissue and vascular channels. Serial blood samples were taken within 10 minutes of fracture and at hourly intervals up to eight hours after fracture. The blood was examined microscopically after the method of Peltier,[15] using Sudan black stain. In addition, lungs were examined histologically for fat emboli.

In 20 out of 25 animals venous blood from the fractured limb showed gross fat embolism when compared with blood from the contralateral intact limb (Fig. 5). When the differences between the number of emboli in control rats and in test animals are analyzed statistically, these differences are found to be significant (>0.01)

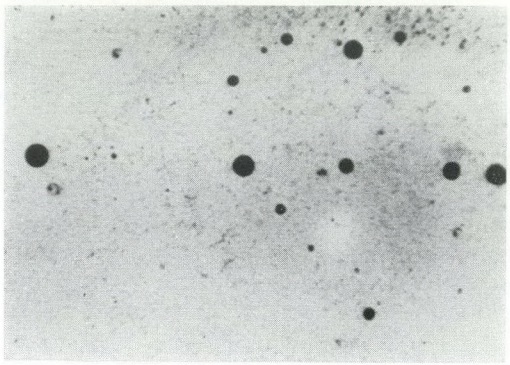


Fig. 5.—Fat emboli in venous blood from fractured limb (Sudan black stain).

at all tested periods. The differences reach an especially high level of significance (>0.001) at intervals of two, three, four and six hours. Scattergrams of the number of emboli plotted against time of blood sampling (Figs. 6 and 7) show these results dramatically.

Histological examination of the lungs correlated well in that emboli numbered 24 to 205 (average 118) per lung section in 19 positive animals. One animal was excluded from these averages since its lung was loaded with fat and therefore the emboli could not be counted. In the five negative rabbits, emboli averaged 28 per section (range 2 to 82). In only one of the negative animals did the number of fat emboli in blood from the intact limb exceed the number in that from the fractured limb. In this animal, fat emboli were also present before fracture, a fact which tends to lessen the significance of this negative result.

Discussion

Earlier experiments in these laboratories clearly established that the fat content of rat bones is sufficient, when introduced intravenously, to produce gross fat embolism.[12] Our observations also confirm that the intravascular fat is not related to hypotension since systemic blood pressures recorded during fracture were seen to rise rather than fall, and induced hemorrhagic shock consistently failed to produce any degree of fat embolism.[13]

Admittedly the tourniquet experiment, herein reported, gives only indirect evi-

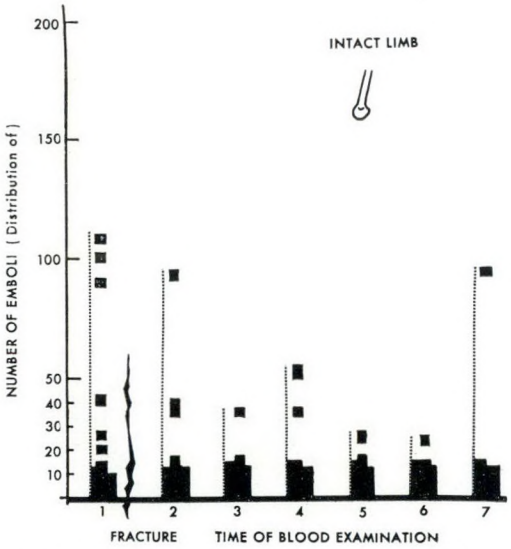


Fig. 6.—Number of emboli from intact limb.

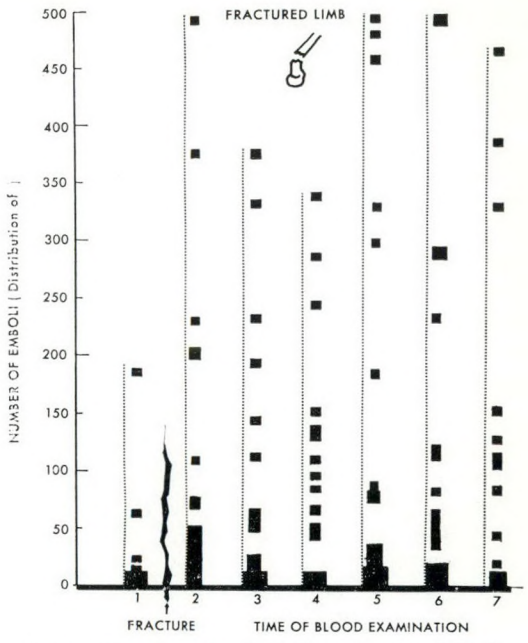


Fig. 7.—Number of emboli from fractured limb.

Bars in Figs. 6 and 7 represent the concentration of emboli appearing (1) immediately before open fracture, (2) within 10 minutes of fracture and (3 to 7) subsequently at hourly intervals.

dence that pulmonary fat emboli originate from the fat of a damaged limb; in this case one which was fractured. The obvious criticism of this experiment is that the tourniquet excludes from the general circulation all other chemical or humoral substances, one of which may be the agent which produces biochemical precipitation of normal blood fat to form emboli.

On the basis of the second experiment, however, one cannot escape the conclusion that fat emboli form within the damaged limb. These emboli appear within 10 minutes of fracture and the speed of their appearance in venous blood from the damaged limb suggests a simple mechanical phenomenon rather than a local biochemical one.

What, then, of the biochemical theory of the pathogenesis of fat emboli? Johnson and Svanborg[7] in an experimental study in rabbits concluded that fat embolism represented one change in a complex system comprising shock, tissue injury and a change in the emulsification of serum lipid. More recently the work of Bergentz[2] and of Adkins and Foster[1] suggests that an agent (thrombin?) produced locally by trauma can cause agglomeration of blood lipids and fat embolism. But these authors as well as Yale and Herrmann[17] accept the fact that there is a primary or contributory mechanical phase in which fat from traumatized areas enters venous channels directly.

It is by no means certain that the two theories are incompatible and this conclusion was anticipated by Swank and Dugger[16] in 1954 and by LeQuire *et al.*[11] in 1959. If one agrees with Adkins and Foster[1] and with Swank and Dugger[16] that hyperlipemia potentiates fat embolism, then a mechanical phase of fat embolism may well be a precursor to the full-blown pathological state precipitated by release of thrombin by damaged tissue. The rarity of the clinical entity and frequency of the pathological one may well be the result of a balance between two factors: first, hyperlipemia due to entry of fat at the fracture site, and second, a tissue substance which agglomerates the fat in blood resulting in fat emboli not only in the lungs but in the systemic circulation as well. If either one of these factors is lacking, the other alone may be insufficient to produce clinical fat

embolism. In this same way, the even more rare occurrence of fat embolism in soft-tissue injury may well be explained.

Summary

Pulmonary fat embolism can be produced in rats by severe fracture and by severe soft-tissue injury and this embolism is enhanced when animals are placed in a warm environment. Lowered blood pressure and vasodilatation do not appear to be factors in this production of fat embolism.

Experiments designed to determine the source of fat in fat embolism led to the inescapable conclusion that there is a mechanical phase in the production of this syndrome, in which depot fat from damaged limbs enters the venous circulation and lodges in the pulmonary capillary bed. This fact, together with evidence from the literature suggesting that a tissue substance is present which agglomerates blood lipids, may account for the sporadic appearance of the clinical syndrome.

The authors are indebted to Miss Joyce Fawsitt for the statistical analyses and to the Department of Medical Illustration for the preparation of the illustrations.

References

1. Adkins, R. B. and Foster, J. H.: Experimental study of the genesis of fat embolism, *Ann. Surg.*, **156:** 515, 1962.
2. Bergentz, S. E.: Studies on the genesis of post-traumatic fat embolism, *Acta Chir. Scand.*, Suppl. **282:** 1, 1961.
3. Downing, T. O.: Fat embolism in goats: 1. Pulmonary fat embolism in goats dying from the effects of massive trauma, U.S. Army Chemical Research and Development Laboratories, Technical Report CRDLR 3106, November 1961.
4. Gauss, H.: The pathology of fat embolism, *Arch. Surg. (Chicago)*, **9:** 593, 1924.
5. Glas, W. W., Musselman, M. M. and Davis, H. L.: The source of fat in fat embolism, *Arch. Surg. (Chicago)*, **71:** 600, 1955.
6. (a) Groskloss, H. H.: Fat embolism, *Yale J. Biol. Med.*, **8:** 59, 1935.
   (b) Groskloss, H. H.: Fat embolism, *Yale J. Biol. Med.*, **8:** 175, 1935.
   (c) Groskloss, H. H.: Fat embolism, *Yale J. Biol. Med.*, **8:** 297, 1936.
7. Johnson, S. R. and Svanborg, A.: Investigations with regard to the pathogenesis of so-called fat embolism: serum lipids and tissue esterase activity and frequency of so-called fat embolism in soft tissue trauma and fractures, *Ann. Surg.*, **144:** 145, 1956.
8. Kane, A. A. *et al.*: Fat embolism: histochemical studies with fluorescent light source and fluorochrome dye (phosphine 3R), *Ann. Surg.*, **153:** 465, 1961.
9. Leblanc, J.: Effect of adrenaline, noradrenaline and chlorpromazine on blood pressure of normal and cold-adapted animals, *Proc. Soc. Exp. Biol. Med.*, **105:** 109, 1960.
10. Lehman, E. P.: Fat embolism, *Int. Surg. Dig.*, **7:** 131, 1929.
11. LeQuire, V. S. *et al.*: A study of the pathogenesis of fat embolism based on human necropsy material and animal experiments, *Amer. J. Path.*, **35:** 999, 1959.
12. Morton, K. S.: Unpublished data.
13. Morton, K. S. and Gurjar, M. P.: Hemorrhagic shock and fat embolism: a clinical and experimental study, *J. Trauma*, **4:** 687, 1964.
14. Peltier, L. F.: Fat embolism: the failure of lipemia to potentiate the degree of fat embolism accompanying fractures of the femur in rabbits, *Surgery*, **38:** 720, 1955.
15. Peltier, L. F.: Fat embolism: the prophylactic value of a tourniquet, *J. Bone Joint Surg.* [*Amer.*], **38A:** 835, 1956.
16. Swank, R. L. and Dugger, G. S.: Fat embolism; clinical and experimental study of mechanisms involved, *Surg. Gynec. Obstet.*, **98:** 641, 1954.
17. Yale, C. E. and Herrmann, L. G.: Pathologic physiology of pulmonary fat embolism: mechanical and chemical alterations with an effective antidote, *Surg. Forum*, **12:** 451, 1961.

Résumé

Les auteurs étudient ici le mécanisme de la production de l'embolisation graisseuse post-traumatique. Ils ont mis au point une technique qui leur permet de reproduire ce phénomène chez le rat de laboratoire. Les animaux furent répartis en cinq groupes. Le premier était, sous anesthésie générale au pentobarbital, soumis à des fractures fermées des deux fémurs et des deux tibias; le deuxième fut soumis au même traumatisme, mais après avoir été préalablement maintenus à une température de 100° F. pendant une heure; le troisième fut traumatisé de façon identique au groupe deux, mais, en outre, reçut une préparation à la chlorpromazine (ceci étant basé sur l'action vaso-dilatatrice de cette drogue); dans le quatrième groupe, on effectua des fractures ouvertes et comminutives des fémurs et des tibias, après réchauffement à 100° F.; enfin le dernier groupe fut soumis à des contusions des parties molles. Les embolies pulmonaires causées par ces divers procédés furent vérifiées à l'aide de coupes congelées des poumons, colorées au Soudan IV et étudiées microscopiquement. Les résultats peuvent être résumés comme suit. L'administration de chlorpromazine n'exerce aucun effet favorisant l'embolisation. Il apparaît comme très probable qu'un facteur mécanique joue ici un rôle important dans la production de ce syndrome, grâce auquel les dépôts graisseux des membres traumatisés peuvent être mobilisés et pénétrer dans la circulation sanguine. De plus, il existe probablement un facteur spécial (une substance d'origine tissulaire) qui cause l'agglutination des lipides sanguins; ce dernier facteur est peut-être le plus important en ce qui concerne l'apparition sporadique de ce syndrome clinique.

EXPERIMENTAL RECONSTRUCTION OF THE CERVICAL ESOPHAGUS WITH A FREE ENTERIC GRAFT*

W. GORDON BEATTIE, M.D., F.R.C.S.[C], F.A.C.S., F. NORMAN BROWN, M.D., F.R.C.S.[C], F.A.C.S., JAMES E. DEVITT, M.D., F.R.C.S.[C], F.R.C.S.(Edin.) and I. J. VOGELFANGER, M.D., M.Sc.D., F.A.C.S., F.I.C.S., *Ottawa, Ont.*

THE bridging of the defect resulting from resection of the cervical esophagus, and/or total laryngopharyngectomy, has taxed the ingenuity of surgeons for the past 75 years. Apart from the occasional *tour de force*[1, 2] before the turn of the century, little was accomplished until Trotter[3] in 1913 outlined his principles of resection and reconstruction of the defect by use of a lateral neck flap. Over the next 30 years many ingenious methods were devised, usually involving staged or multiple operative procedures. One of the best of these proved to be the operation developed by Wookey of Toronto.[4, 5] Despite the safety and effectiveness of this procedure, it was characterized by prolonged morbidity and the need for difficult nursing care between operations.[6]

The continuing challenge led others[7-9] to try single-stage procedures for resection and reconstruction using pedicled transplants of large and small bowel passing through the intervening chest cavity either in front of or behind the sternum or via the posterior mediastinum. These formidable procedures resulted in a high mortality in the fragile, poorly nourished and often elderly patients on whom these were undertaken. Owing to the precarious and somewhat unpredictable blood supply through the pedicle, an anastomotic leak led to death or fistula formation in a significant number of these patients.[8]

In an endeavour to simplify the procedure Rob and Bateman[10] in Great Britain and Edgerton[11] in the United States attempted a primary reconstruction by means of tantalum-supported fascia lata or dermis grafts. Despite the initial enthusiasm, leaks and fistulas occurred early in the postoperative course and stenosis was almost invariably a late complication. Numerous modifications of this method have been attempted, some of which have similar defects and others which have not been fully developed.[12] An occasional successful replacement has been carried out, using the larynx.[13]

Progress beyond this point had to await developments in the field of organ transplantation. With an awakening interest in organ grafting, technical problems of transplantation became important. With increasing skill, surgeons adapted finer and finer suture material to smaller and smaller vessels, leading to microsurgical techniques involving operating microscopes.[14, 15]

Although satisfactory in certain situations, the limitations of the unassisted human hand, especially in the field of venous anastomosis, became evident to many investigators.

The timely development of the Canadian stapler (Fig. 1) with its automatic everter (Fig. 2) by Vogelfanger and his associates,[16-19] in conjunction with the Department of Mechanical Engineering of the National Research Council, provided this assistance especially in the low-pressure venous system. Seidenberg *et al.*[20] attempted to reconstruct the cervical esophagus in the dog with free bowel transplants but had considerable difficulty handling the small fragile vessels in the mesentery. In an attempt to overcome this technical problem the authors used the Vogelfanger-NRC stapler.

This is a report of the method and materials of an investigation during a two-year period from 1961 to 1963.

---

*From the Department of Experimental Surgery, Ottawa Civic Hospital; Department of Anatomy, University of Ottawa, and the National Research Council of Canada.

Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Quebec, January 1964, and at the Janes Surgical Society, Edinburgh, October 1964.

Supported by grants from the Medical Research Council, the Ontario Cancer Foundation and the Department of Medical Education and Research of the Ottawa Civic Hospital.

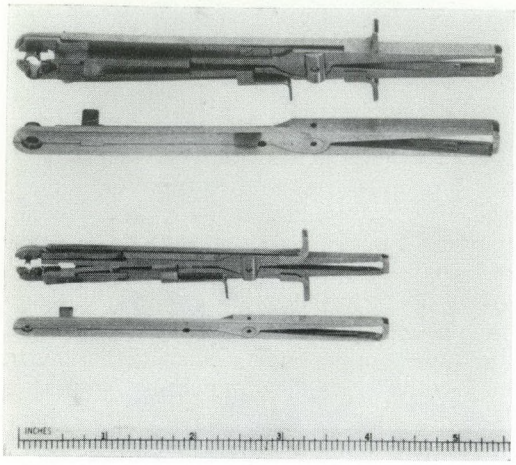


Fig. 1.—(Top) Vogelfanger-NRC stapler, Mark IV. (Bottom) Vogelfanger-NRC stapler, Mark V.

Methods and Materials

Mongrel dogs, weighing 25 to 35 kg., were placed on a low residue diet for five days with water only for 24 hours before the operation.

Two surgical teams simultaneously prepared the recipient site in the neck and the intestinal donor segment in the abdomen. The lumen of the intestine was washed with 1% neomycin and the carefully preserved segmental vessels were perfused with heparin 0.01%. A single layer end-to-end anastomosis reconstituted the intestinal tract. The superior thyroid artery and anterior facial vein were cleared. Approximately 4 cm. of the cervical esophagus was resected to receive the graft. The prepared vessels in the neck were joined to their respective enteric graft vessels using the Vogelfanger-NRC stapler (Fig. 3). The Mark IV instrument was used for the vessels over 2 mm. (usually veins), and the Mark V for vessels under 2 mm. (arteries).

After the revascularization, the graft was implanted into the esophageal defect and anastomosed end to end with the esophagus with a single layer of interrupted 000 silk.

Postoperatively, all animals were kept on intravenous fluids for 48 hours. Oral feeding was begun with fluid, progressing through semi-solids to full kennel rations over a period of 10 to 14 days. If a complication occurred, the dogs were esophagoscoped and the specimen was then removed for study. If no complication developed, the dogs were followed up for intervals of up to six months. At the end of a varying period of observation, each dog underwent a cine-esophageal barium study, esophagoscopy and excision of the specimen, including graft vessels and the adjacent portion of the esophagus above and below the graft. Angiography of the graft vessels was carried out before gross and microscopical examination.

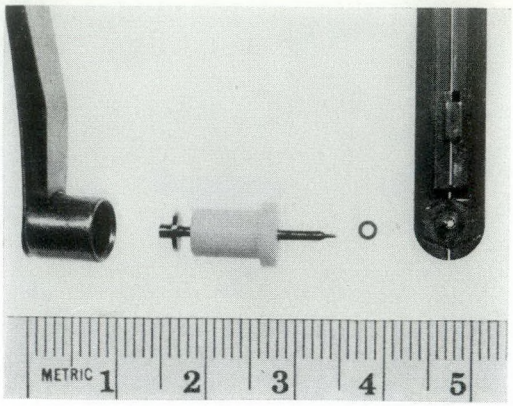
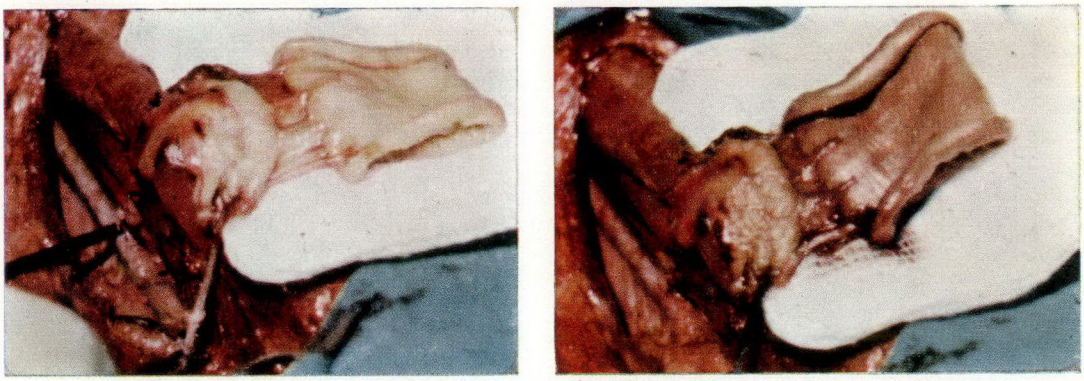


Fig. 2.—The automatic everter disassembled.

The project was undertaken in three stages: (1) Orientation and instrument development. (2) Jejunal transplantation. (3) Colon transplantation.

Observations and Results

Little difficulty was met with in isolating and perfusing the jejunal or colonic segments for transplantation into the defect in the neck. The vessels were anastomosed first before implanting the graft. When the hemostatic clamps were released, the vessels in the graft immediately started to pulsate, the graft became pink and its edges bled. An adjustment of blood flow seemed to take place over the next 10 to 15 minutes, judged by the colour and spasticity of the graft and the amount of mucus it produced. If the flow was adequate, the graft stayed pink and soft, and little mucus was secreted. This almost invariably heralded a successful final result. On the other hand, if the flow was not satisfactory, the graft became purplish, hard and spastic and produced large quantities of mucus. When this occurred, the transplant usually failed.



(a) (b)

Fig. 3.—(a) Artery and vein anastomosed and the hemostatic clamp on artery. The graft is pale and the vessels are collapsed. (b) The arterial clamp has been removed. The graft is pink and the vessels are full.

A discrepancy between the size of the vessels of the graft and those of the recipient was the major problem. The anastomosis would fail if the inequality were greater than 0.25 mm. in vessels under 2 mm. and greater than 0.5 mm. in those over 2 mm.

The cine-esophageal barium study (Fig. 4) on the only survivor free of complications after a jejunal transplant revealed marked spasm, a finding also reported by Hopkins and Bernatz.[21] There was little or no spasm in any of the colon transplants on cine-barium studies (Fig. 5). These structures appeared to act as admirable conduits in which no evidence of peristaltic activity was detected.

The angiogram (Fig. 6) of a colon graft

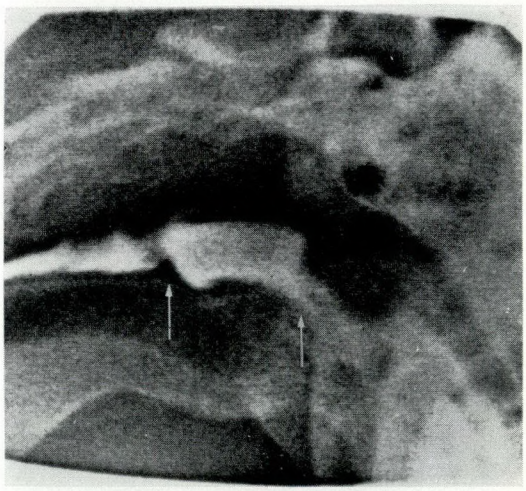


Fig. 5.—Colon graft outlined by arrows. No spasm.

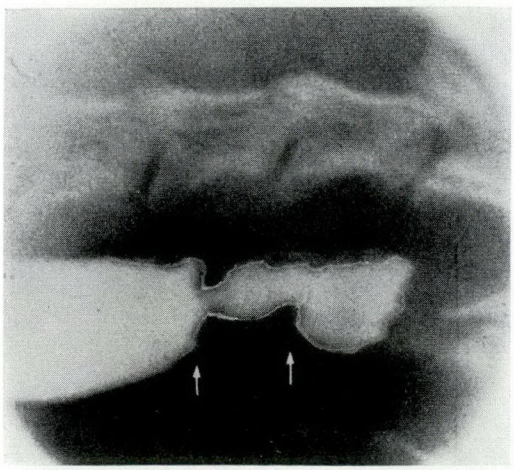


Fig. 4.—Jejunal graft outlined by arrows. Note marked spasm.

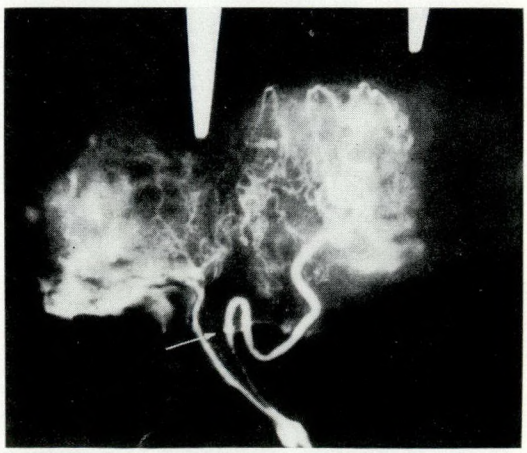


Fig. 6.—Angiography of colon graft six months after implant. Arrow points to staples and the mosquito tips outline the graft.

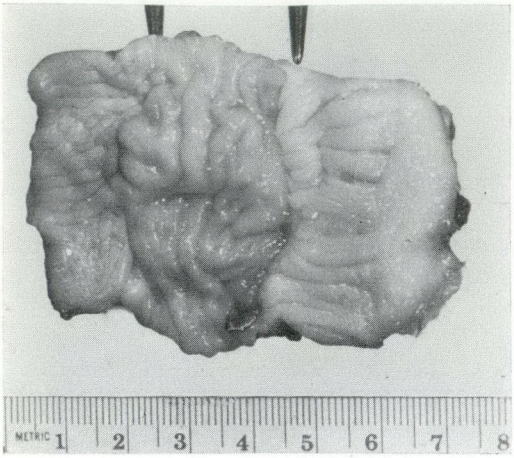


Fig. 7.—Colon graft six months after implantation. Opened specimen.

six months after operation revealed that the artery was patent, with a branch going to the graft and one to the cephalad part of the esophagus. The fine tantalum staples can be seen. It is interesting to observe that the branch artery supplies the graft completely with little if any collateral supply across the junction of the transplant and esophagus.

The opened specimen (Fig. 7), in which the dark transverse folds of the transplant are in sharp contrast to the longitudinal light-coloured folds of the esophagus, reveals no stenosis.

In the microscopical section (Fig. 8), the apposition of the columnar and stratified epithelium can be easily seen. Similarly, at the junction of the striated muscle of the esophagus and the smooth muscle of the bowel, there is a minimum of fibrous tissue indicating the line of fusion.

TABLE I.—Cervical Esophageal Reconstruction: Initial Investigation—17 Dogs

Anatomical assessment	2
Perfusion	4
In situ vascular anastomosis	11

The initial phase of this investigation is summarized in Table I. This series of dogs was used to establish anatomical parameters (in particular measurement of vessel size), to learn perfusion techniques and to test the developing Mark V stapler on *in situ* transplants.

The results obtained with jejunal transplants are summarized in Table II. The mesenteric vessels were small and fragile. Some failures were due to this fact, but most were due to disparity in size and occasionally to instrument

Fig. 8.—Microphotograph of junction of colon graft and esophagus.

TABLE II.—Cervical Esophageal Reconstruction: Jejunal Transplants—16 Dogs

Completed operation		2
Uncomplicated recovery	1	
Complications	1	
Not completed		14

TABLE III.—Cervical Esophageal Reconstruction: Colon Transplants—16 Dogs

Completed operation		8
Uncomplicated recovery	4	
Complications	4	
Not completed		8

failure. When a vessel anastomosis was unsuccessful, the segment of bowel was not implanted. When the graft appeared to be well vascularized, the reconstruction of the esophageal defect was completed.

Failure of the jejunal graft was due to ischemic necrosis at the suture line with associated leak, abscess formation and subsequent fistula formation. This would develop within 7 to 10 days of the operation, although one initially satisfactory transplant became stenosed and a fistula developed two weeks after the operation. When at six weeks an autopsy was performed on the dog, a fibrosed graft was found with thrombosed vessels. The only dog that survived without complications was followed up for three months and then sacrificed. The graft had a good lumen and there was no evidence of stenosis. Despite this fact, the cine-barium studies had previously revealed marked spasm and the dog failed to maintain his weight after operation.

The colon grafts proved to be more satisfactory technically since the vessels were somewhat larger and less fragile (Table III). Despite this, technical difficulties were experienced with the vessels, mainly due to discrepancy in size. This led to abandoning the implant in eight dogs. Of the eight dogs in which the procedure was completed, four developed complications, three had fistulas within two weeks. At autopsy, the grafts were necrotic and the vessels thrombosed. One dog made an uncomplicated recovery but at esophagoscopy had a stenosis. At autopsy three months after the operation, the graft was narrowed and scarred and only a few small islands of colonic mucosa remained. The artery was narrowed and the vein thrombosed. The four dogs that had an uncomplicated recovery had a good lumen on cine-barium study and esophagoscopy. At autopsy there was no stenosis and the vessels were patent. The longest elective survival was six months.

Summary and Conclusions

An experimental method for the reconstruction of the cervical esophagus in the case of defects after resection has been described, using free enteric transplants from jejunum and colon in mongrel dogs. Revascularization of the transplant was accomplished by joining the respective segmental mesenteric vein and artery to the anterior facial vein and superior thyroid artery in the neck, utilizing the Vogelfanger-NRC stapler. The jejunum was unsatisfactory as a graft source because the small vessels were very fragile, the discrepancy in size between graft vessels and recipient vessels was too great, and function of the surviving graft was inadequate due to spasm. The colon segment was more readily handled and proved to be an acceptable conduit. This experience provides further support for the conclusion that the revascularized transplant of colon is superior to jejunum for bridging an alimentary tube defect in the neck. Clinical application awaits further study.

The authors wish to thank Dr. D. C. MacPhail, Director, Mr. S. Connock, Head, Instruments and Control System Laboratory, Mr. A. Smialowski, Engineer with the National Research Council, and Mr. W. J. Watson, designer. The assistance of the following is also greatly appreciated: Professor J. Auer, Department of Anatomy, University of Ottawa; Dr. M. Klotz, Head of the Department of Pathology; Dr. D. Cockburn, Head of the Department of Diagnostic Radiology, and Mr. M. Smith, Department of Photography, all of the Ottawa Civic Hospital.

References

1. Czerny, V.: Resection des Oesophagus, *Zbl. Chir.*, **4:** 433, 1877.
2. Mikulicz, J.: Ein Fall von Resection des carcinomatösen Oesophagus mit plastischem Ersatz des excidierten Stückes, *Prag. Med. Wschr.*, **11:** 93, 1886.
3. Trotter, W.: The Hunterian lectures on the principles and techniques of the operative treatment of malignant disease of the mouth and pharynx, *Lancet*, **1:** 1075, 1913.
4. Wookey, H.: The surgical treatment of carcinoma of the pharynx and upper esophagus, *Surg. Gynec. Obstet.*, **75:** 499, 1942.

5. WOOKEY, H.: Surgical treatment of carcinoma of the hypopharynx and oesophagus (Hunterian lecture), *Brit. J. Surg.*, **35:** 249, 1948.
6. COWAN, R. J. AND MACDOUGALL, J. A.: Reconstruction of the pharynx following laryngo-pharyngo-oesophagectomy, *Plast. Reconstr. Surg.*, **21:** 362, 1958.
7. YUDIN, S. S.: The surgical construction of 80 cases of artificial esophagus, *Surg. Gynec. Obstet.*, **78:** 561, 1944.
8. GOLIGHER, J. C. AND ROBIN, I. G.: Use of the left colon for reconstruction of the pharynx and oesophagus after pharyngectomy, *Brit. J. Surg.*, **42:** 283, 1954.
9. MUSTARD, R. A. AND IBBERSON, O.: Carcinoma of the esophagus; a review of 381 cases admitted to Toronto General Hospital 1937-1953 inclusive, *Ann. Surg.*, **144:** 927, 1956.
10. ROB, C. G. AND BATEMAN, G. H.: Reconstruction of the trachea and cervical oesophagus; preliminary report, *Brit. J. Surg.*, **37:** 202, 1949.
11. EDGERTON, M. T.: One-stage reconstruction of the cervical esophagus or trachea, *Surgery*, **31:** 239, 1952.
12. SCHOBINGER, R.: The use of platysma and nylon tubing in the reconstruction of the cervical esophagus in dogs, *Plast. Reconstr. Surg.*, **23:** 36, 1959.
13. WOODS, L. P. AND ROSENFELD, L.: Laryngeal substitution for the cervical esophagus: a case report, *Ann. Surg.*, **157:** 653, 1963.
14. JACOBSON, J. H., 2ND AND SUAREZ, E. L.: Microsurgery in anastomosis of small vessels, *Surg. Forum*, **11:** 243, 1960.
15. JACOBSON, J. H., 2ND AND SUAREZ, E. L.: Progress in cardiovascular surgery; microvascular surgery, *Dis. Chest*, **41:** 220, 1962.
16. VOGELFANGER, I. J. AND BEATTIE, W. G.: A concept of automation in vascular surgery: a preliminary report on a mechanical instrument for arterial anastomosis, *Canad. J. Surg.*, **1:** 262, 1958.
17. VOGELFANGER, I. J. *et al.*: The problem of small vessel anastomosis, *Surgery*, **52:** 354, 1962.
18. SCOBIE, D. H., SCOBIE, T. K. AND VOGELFANGER, I. J.: Venous autografts, *Canad. J. Surg.*, **5:** 471, 1962.
19. WADDELL, W. G. *et al.*: Venous valve transplantation, *Arch. Surg. (Chicago)*, **88:** 5, 1964.
20. SEIDENBERG, B. *et al.*: Immediate reconstruction of the cervical esophagus by a revascularized isolated jejunal segment, *Ann. Surg.*, **149:** 162, 1959.
21. HOPKINS, D. M. AND BERNATZ, P. E.: Experimental replacement of the cervical esophagus, *Arch. Surg. (Chicago)*, **87:** 265, 1963.

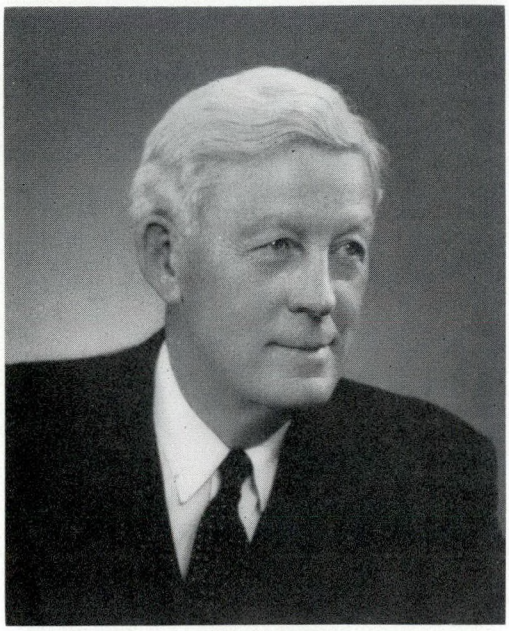
RÉSUMÉ

De très nombreuses variantes ont été proposées en ce qui concerne les plasties œsophagiennes, et les auteurs ont procédé à une étude expérimentale de ce problème. A cette fin, des chiens bâtards, pesant entre 25 et 35 kg., furent utilisés. Les animaux furent préparés par une diète pauvre en résidus pendant cinq jours, et mis à la diète à l'eau 24 heures avant l'intervention. Deux équipes opératoires travaillèrent simultanément, l'une sur l'abdomen et l'autre sur la région œsophagienne. Dans la cavité péritonéale, on procéda à une résection intestinale partielle, et le segment réséqué, qui était destiné à fournir le matériel de plastie, fut soigneusement disséqué, en conservant ses vaisseaux. Immédiatement après excision, l'intestin fut lavé avec de la néomycine et les vaisseaux perfusés à l'aide d'une solution d'héparine. La continuité de la lumière intestinale fut rétablie par une anastomose bout-à-bout. Dans la région du cou, on disséqua l'œsophage et on en réséqua approximativement 4 cm. Les vaisseaux furent soigneusement conservés et suturés aux vaisseaux intestinaux; cette délicate opération fut effectuée grâce à l'agraffeur Vogelfanger, récemment developpé en coopération avec le Conseil Médical de la Recherche du Canada. Après revascularisation, le greffon intestinal fut alors adapté et suturé bout-à-bout aux segments œsophagiens. Post opératoirement, les animaux furent maintenus à la diète totale pendant 48 heures. Les greffons utilisés provenaient soit de la portion jéjunale du grêle, soit du côlon. Les chiens furent très soigneusement étudiés au point de vue anatomique et microscopique; des contrôles radiologiques furent effectués et la vascularisation étudiée par ciné-angiographie. Les résultats ont été très encourageants avec la transplantation du côlon.

EDITORIAL TRIBUTE

ROBERT MEREDITH JANES

Scholar, Teacher, Editor and Surgeon



Dr. R. M. Janes

FROM the inception of the *Canadian Journal of Surgery* in 1957 until he asked to be replaced as Chairman of the Editorial Board, Dr. R. M. Janes has served in this demanding and significant appointment in all weathers and in all fortunes. Few except the editors and those who have served on the Board can appreciate to the full that whatever success the Journal has achieved has come in large measure through his faith, wisdom and diligence. Without question this particular service to the profession is performed best when it is performed as a labour of love.

Dr. Janes was Professor and Head of the Department of Surgery of the Faculty of Medicine of the University of Toronto and Surgeon-in-Chief of the Toronto General Hospital from 1947 until 1957. During this period, many important developments occurred, both in the Department of Surgery and at the hospital. At the end of World War II, a large number of young Canadian doctors returned from service in the Armed Forces and sought graduate training in surgery. To provide them with the training which they deserved, the graduate training program in surgery, which had been initiated by Dr. Gallie, was rapidly expanded to include all the teaching hospitals of the University, Sunnybrook Hospital (DVA) and, to a limited degree, certain large non-teaching hospitals. At the same time, the training programs for the sub-specialties within the department were developed and expanded.

At the Toronto General Hospital Dr. Janes was Chairman of the committee which planned the major post-war building program that resulted in the large new centre block and its associated structures.

During this busy period he was also giving his time unstintingly to the Royal College of Physicians and Surgeons of Canada. He served on the Council of the College, on many committees, and from 1951 until 1953 was Vice-President, Surgery, and from 1955 until 1957 was President of the College. He still serves as a corresponding member of the Policy Committee, which consists of the President and recent Past-Presidents.

Since his retirement from the Chair of Surgery to the position of Emeritus Professor of Surgery in the University of Toronto, his wisdom and mature judgment have been called upon freely by a number of organizations. He has been Consultant Surgeon to the Humber Memorial Hospital, Toronto, and several of the members of the Department of Surgery of that institution had taken their training in surgery under his guidance. He has been a Consultant in the planning of the North York Hospital and Chairman of CARE of Canada. In 1958, he had the great distinction of being appointed as Sims Travelling Professor and in this capacity he and Mrs. Janes made an extended tour of the medical schools in South and Central Africa. He made many friends in Africa, where the breadth of his knowledge of surgery, as well as his interest in and experience in teaching, both graduate and undergraduate, were greatly appreciated. This journey to Africa gave him an excellent opportunity to exercise his hobby of photography, in which he is an acknowledged expert.

The surgeons who took training under his direction at the University of Toronto formed the Janes Club, which meets annually for a scientific session and to renew old acquaintances. Dr. Janes has justifiable pride in the high professional standard of these surgeons,

some 125 in number, who conduct their practices in centres scattered from Vancouver to St. John's, Newfoundland, and to some extent outside Canada. In 1964 Dr. Janes and members of the Club paid a visit to Great Britain and were royally entertained by Dr. Janes' friends among the leading surgeons of Glasgow, Edinburgh and London.

Dr. Janes has continued to be active in private practice until very recently and his retirement is much regretted by his patients, a large number of physicians who for many years have trusted him with their difficult surgical problems, and by his confreres in surgery.

The choice of Dr. Janes' successor as Chairman of the Editorial Board of this Journal was made somewhat easier because he made known his intention to withdraw more than a year ago and because, close at hand, a distinguished surgeon with a deep and abiding interest in medical communications was found, willing to serve. Thus, with pride and satisfaction the members of the Editorial Board of the *Canadian Journal of Surgery* welcome as their new chairman, Dr. Frederick G. Kergin, Professor and Head, Department of Surgery, University of Toronto.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Acute Injuries of the Head. Their Diagnosis, Treatment, Complications and Sequels. 4th ed. G. F. Rowbotham. 584 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1964. $16.25.

Advances in Surgery. Vol. 1. Edited by Claude E. Welch and six associate editors. 370 pp. Illust. Year Book Medical Publishers, Inc., Chicago, Ill., 1965. $13.75.

Burns. A Symposium. Edited by Leon Goldman and Richard E. Gardner. 191 pp. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1965. $9.25.

The Coronary Arteries. Arteriography, Microanatomy, and Pathogenesis of Obliterative Coronary Artery Disease. William F. M. Fulton. 354 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1965. $24.50.

The Craft of Surgery. Vol. I. Edited by Philip Cooper. pp. 1-819. Illust. Little, Brown and Company, Boston; J. B. Lippincott Company of Canada Ltd., Montreal, 1964. $4.50.

The Craft of Surgery. Vol. II. Edited by Philip Cooper. pp. 823-1510. Illust. Little, Brown and Company, Boston; J. B. Lippincott Company of Canada Ltd., Montreal, 1964. $4.50.

Idiopathic Hypertrophic Subaortic Stenosis. From the Cardiology Branch and Clinic of Surgery, National Heart Institute, Bethesda, Maryland. American Heart Association Monograph No. 10. 213 pp. Illust. Distribution Department, American Heart Association, Inc., New York, 1964. $2.50.

Manuel de Traumatologie (Membres-Rachis-Ceintures). Georges Rieunau. 246 pp. Illust. Masson & Cie, Paris, 1964. 50 NF. $10.00 (approx.).

Modern Ophthalmology. Vol. 1. Basic Aspects. Edited by Arnold Sorsby. 531 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1963. $29.25.

Neoplastic Disease at Various Sites. General Editor: D. W. Smithers. Vol. V. Tumours of the Kidney and Ureter. Edited by Sir Eric Riches. 416 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1964. $18.00.

Plastic Surgery. Richard Battle. 416 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1964. $22.00.

Plastic Surgery Nursing. Mair M. Jenkins. 128 pp. Illust. The Macmillan Company of Canada Limited, Toronto, 1964. $3.00.

Technique of Internal Fixation of Fractures. M. E. Müller. M. Allgöwer and H. Willenegger, with other contributors. Revised for the English edition by G. Segmuller. 271 pp. Illust. Springer-Verlag New York, Inc., New York, 1965. $20.00 U.S.

Traité de Thérapeutique Chirurgicale. Edited by J. Senèque. Tome I. Chirurgie orthopédique et traumatologie des membres-rachis-bassin. R. Judet and J. Judet et al. 733 pp. Illust. Masson & Cie, Paris, 1964. 156 F. $31.20 (approx.).

Traité de Thérapeutique Chirurgicale. Edited by J. Senèque. Tome IV. Affection du foie, des voies biliaires de la rate et du pancréas. M. Mercadier, avec la collaboration de J.-M. Brisset, P. Hautefeuille, P. Vayre. 754 pp. Illust. Masson & Cie, Paris, 1964. 156 F. $31.20 (approx.).

The Year Book of General Surgery (1964-1965 Year Book Series). Edited by M. E. De Bakey. 637 pp. Illust. Year Book Medical Publishers, Inc., Chicago, Ill., 1964. $9.35.

The Royal College of Physicians and Surgeons of Canada

NEWSLETTER

1. 1965 ANNUAL MEETING

The 1965 Annual Meeting of the College held recently in Toronto set new records for attendance at these meetings, with a registration of over 1600, including over 1100 Fellows and 240 certificated specialists. More than 200 new Fellows were presented at the annual convocation, an impressive ceremony held in the Convocation Hall of the University of Toronto. The academic procession included six past-presidents of the College as well as official representatives from six sister Colleges in the United States and the United Kingdom. A total of 131 papers made up the scientific program, including those of the guest lecturers and the medallists. A session of general interest in the field of the neurological sciences, which represented an attempt by the Committee on Arrangements for the Annual Meeting to extend the appeal of the scientific program to those in the subspecialties of medicine and surgery, proved highly successful, with attendance at this session ranging as high as 300. Colour television, sponsored by Smith, Kline and French, was once again a popular addition to the scientific sessions. More than 900 attended the President's Reception and Annual Dinner on Saturday evening and heard a stimulating address by Mr. Arnold Edinborough, Editor of *Saturday Night*, on the topic "The Public Image of the Medical Profession".

Once again, the thanks of the College are due to a great many people for the interest and efforts which make successful meetings possible. It is not possible to name them all but particular thanks are due to the three program chairmen: Dr. H. Garfield Kelly in Medicine, Dr. Norman C. Delarue in Surgery and Dr. Douglas E. Cannell in Obstetrics and Gynecology; to Dr. Delarue who assumed also the responsibility of Chairman of the Committee on Local Arrangements, and to Mrs. Esther Mills, wife of Dr. Frank Mills, who chaired the Ladies' Committee.

2. SCHEDULE OF FUTURE ANNUAL MEETINGS

Sites and dates for future Annual Meetings of the College have been established as follows:

1966—Queen Elizabeth Hotel, Montreal—January 20-22

1967—Chateau Laurier Hotel, Ottawa—January 19-21

1968—Royal Alexandra Hotel, Winnipeg—January 25-27

1969—Royal York Hotel, Toronto—January 22-24

1970—Chateau Frontenac Hotel, Quebec—January 23-25

3. 1965 REGIONAL MEETING

The 1965 Regional Meeting of the College will be held at the Admiral Beatty Hotel, Saint John, N.B., on Thursday, Friday and Saturday, October 7-9, 1965. Notices and invitations to attend have been mailed to all Fellows and Certificants of the College in the Atlantic Provinces, the region which the meeting is designed primarily to serve. However, any Fellow or Certificant throughout the rest of Canada will be most welcome. Inquiries may be directed to Dr. W. D. Miller, Chairman, Committee on Local Arrangements, 115 Hazen Street, Saint John, N.B.

4. FUTURE REGIONAL MEETINGS

The Council has agreed that, commencing in 1966, the College will embark on a program of two Regional Meetings per year. Future Regional Meetings have been scheduled as follows:

1966—Saskatoon and Sudbury
1967—Calgary and Chicoutimi
1968—Windsor and Halifax

(Continued on page 231)

CANADIAN JOURNAL OF SURGERY

All communications concerning this Journal should be marked "Canadian Journal of Surgery" and addressed to the Editor, C.M.A. Publications, at C.M.A. House, 150 St. George St., Toronto 5.

The Journal is published quarterly. Subscription is $10 per year ($5 per year for trainees in surgery), and starts with the January issue of each year. Single copies are $2.50 each, payable in advance. (It would be greatly appreciated if subscribers would please add bank exchange to their cheques.)

INSTRUCTIONS TO CONTRIBUTORS

Manuscripts

Manuscripts **in duplicate** of original articles, case reports, and other contributions should be forwarded with a covering letter requesting consideration for publication in the *Canadian Journal of Surgery*. Acceptance is subject to the understanding that they are submitted solely to this Journal, and will not be reprinted without the consent of the author and the publishers. Acceptance or rejection of contributions will be determined by the Editorial Board. As space is available, a limited number of case reports will be published. Articles should be typed on one side only of unruled paper, double-spaced, and with wide margins. The author should always retain a carbon copy of material submitted. Every article should contain a summary of the contents. The Concise Oxford Dictionary will be followed for spelling. Dorland's American Medical Dictionary will be followed for scientific terminology. The Editorial Board reserves the right to make the usual editorial changes in manuscripts, including such changes as are necessary to ensure correctness of grammar and spelling, clarification of obscurities or conformity with the style of the *Canadian Journal of Surgery*. In no case will major changes be made without prior consultation with the author. Authors will receive galley proofs of articles before publication, and are asked to confine alterations of such proofs to a minimum.

Reprints

Reprints may be ordered on a form which will be supplied with galley proofs. It is important to order these before publication of the article, otherwise an extra charge for additional type-setting will be made.

References

References should be referred to by numerals in the text. They should include in order: the author's name and initials in capitals; title of the article; abbreviated journal name; volume number, page number and year. The abbreviations of journal names should be those used by the National Library of Medicine, Washington, D.C., as published in *Index Medicus*. References to books should include in order: author's name and initials; title of book; number of edition (e.g., 2nd ed.); title of publishing house; city of publication; year of publication; page number if a specific reference.

Illustrations

A reasonable number of black-and-white illustrations will be reproduced free with the articles. Colour work can be published only at the author's expense. Photographs should be glossy prints, unmounted and untrimmed, preferably not larger than 10″ x 8″. Prints of radiographs are required and *not the originals*. The magnification of photomicrographs must always be given. Photographs must not be written on or typed on. An identifying legend may be attached to the back. Patients must not be recognizable in illustrations, unless the written consent of the subject for publication has been obtained. Graphs and diagrams should be drawn in India ink on suitable white paper. Lettering should be sufficiently large that after reduction to fit the size of the Journal page it can still be read. Legends to all illustrations should be typed separately from the text and submitted on a separate sheet of paper. Illustrations should not be rolled or folded.

Language

It should be clearly understood that contributors are at full liberty to submit articles in either English or French, as they please. Acceptance will be quite independent of the language of submission. If the contributor wishes, he may submit an informative summary of not more than 300 words in the language other than that in which he has submitted the article. For example, an article in English must carry an English summary and may, if the author wishes, carry a more detailed summary in French.

(Continued from page 229)

5. AWARD OF 1965 ROYAL COLLEGE TRAVEL FELLOWSHIPS

The Committee on Administration of the Educational Endowment Fund of the College has selected the following Fellows to receive Royal College Travel Fellowships, in the amount of $1000 each, to assist them in undertaking additional postgraduate studies:

Dr. John Edward Campbell, Montreal
Dr. William Allan Mahon, Edmonton
Dr. Emile Marcotte, Montreal
Dr. Atm Prakash, New Delhi

6. MERCK, SHARP & DOHME TRAVEL FELLOWSHIP

The Committee on Administration of the Educational Endowment Fund has awarded the 1965 Merck, Sharp and Dohme Travel Fellowship to Dr. Paul Benoit, Research Fellow in the Hematological Research Laboratory of Sainte-Justine Hospital, Montreal. This Fellowship, amounting to $6000, will enable Dr. Benoit to spend approximately 16 months in clinical immunohematological research at the Hôpital Saint-Louis in Paris followed by visits to other centres in the United Kingdom and the United States.

7. MODIFICATION OF THE TRAINING REQUIREMENTS FOR THE FELLOWSHIP EXAMINATION IN SURGERY MODIFIED FOR NEUROSURGERY

Commencing in 1968, candidates for the Fellowship examination in Surgery modified for Neurosurgery will be required to have fulfilled a training program totalling six years, which must include at least one year of approved resident training in General Surgery, and at least two and a half years of approved resident training in clinical Neurosurgery. Candidates desiring further information concerning the new training requirements in this specialty should write to the Secretary of the College, 74 Stanley Avenue, Ottawa 2, Ont.

W. Gordon Beattie, F.R.C.S.[C],
Honorary Assistant Secretary,
March 12, 1965.

FORTHCOMING MEETINGS

THE SOUTH WESTERN ONTARIO SURGICAL ASSOCIATION

The Annual Meeting of the South Western Ontario Surgical Association will be held in the Busby Memorial Auditorium, Victoria Hospital, London, Ont., on Wednesday, November 3, 1965.

For further details, please write to Dr. T. D. McLarty, Program Chairman, Surgery Office, Victoria Hospital, London, Ont.

THE CANADIAN SOCIETY FOR THE STUDY OF FERTILITY

The Twelfth Annual Meeting of the Canadian Society for the Study of Fertility will take place at the Nova Scotian Hotel, Halifax, N.S., on June 9 and 10, 1965.

For further information, please write to the Secretary, Dr. John R. O'Brien, 3550 Cote des Neiges Road, Suite 680, Montreal, Que.

NOTICES

JAMES IV SURGICAL ASSOCIATION AWARD

Dr. R. A. Macbeth, Professor of Surgery at the University of Alberta, Edmonton, and a member of the Editorial Board of the Journal, has been awarded a surgical travellership by the James IV Surgical Association. Commencing April 1, 1965, Dr. Macbeth will spend approximately two months in Great Britain and the Scandinavian countries.

SMALL-BOWEL ULCER REGISTRY

A central registry for small-bowel ulcers has recently been set up at the Jewish Hospital of Brooklyn. It is hoped that all cases of small-bowel ulcer, regardless of medication or underlying disease, will be reported to this agency, from which appropriate forms can be obtained. Periodic reports will be issued and pertinent literature and pathological material will be made available to those interested.

For further information, please write to the Small-Bowel Ulcer Registry, The Jewish Hospital of Brooklyn, 555 Prospect Place, Brooklyn, N.Y. 11238, U.S.A.

BOOK REVIEWS

(See also pages 136, 187, 200 and 213)

EXPERIMENTAL SURGERY. Including Surgical Physiology. 5th ed. J. Markowitz, J. Archibald and H. G. Downie. Foreword by Frederick G. Kergin. 659 pp. Illust. The Williams & Wilkins Company, Baltimore; Burns & MacEachern Ltd., Don Mills, Ont., 1964. $13.00.

This fifth edition of "Experimental Surgery" presents a concise and systematic review of surgical physiology and experimental surgical techniques, and relates these to the clinical practice of surgery. The book will be of particular value to surgical residents because it traces the evolution of many commonly employed procedures from the original experimental work to their application in man. The numerous historical notes and quotations from key articles are supported by excellent illustrations and well-chosen references.

The chapters on gastrointestinal, thoracic and vascular surgery are particularly good and can be recommended even to those who do not intend to carry out the experimental procedures described. The chapter on transplantation of organs, as those in other textbooks at the present time, might have been improved by outlining the history of transplantation and by defining the terms that are commonly used by workers in this field.

The authors are to be congratulated on producing a wealth of information, nicely written and presented, in a reasonably sized book.

DIAGNOSTIC UROLOGY. Edited by James F. Glenn. 415 pp. Illust. Hoeber Medical Division, Harper & Row, Publishers, Inc., New York 16, N.Y., 1964. $13.50.

This authoritative book on "Diagnostic Urology" cannot be recommended too highly. Usually in a book with multiple authorship the standard of presentation from chapter to chapter is uneven. The editor is to be commended for maintaining an extremely high standard throughout.

Each chapter is laid out in a very clear manner with headings which outline completely the subject matter of the chapter. Each chapter is also provided with a list of references which are uniformly helpful.

The book achieves a very satisfactory amalgam of the old and the new in diagnostic urological methods. So far as the old is concerned, it very properly begins with a discussion of history-taking and physical examination. The editor himself deals with this opening chapter and the fundamental remarks made here require notice by all — not only those in training but those of us who are practising the specialty day by day. The old as represented by cystoscopic methods, upper urinary tract urography, and cystourethrography are very well dealt with, but particular mention has to be made of the somewhat newer urological studies which are now available. Quite outstanding are chapters on retroperitoneal contrast studies, radioisotope renography, reno-radio-isotope evaluation of function and scanning, aortography and lymphangiography. All of these are dealt with by masters in the field, and the advantages and disadvantages of these methods are clearly stated.

Urology, like other specialties, has its Cinderella features. In urology, the Cinderellas are the diagnosis of male infertility and the problem of intersexuality. Both of these subjects are most adequately dealt with by urologists who are obviously enthusiasts in their fields.

Because each chapter is authoritatively dealt with, this book can be strongly recommended to everyone in training in urology and as a handbook for those practising urology. The foreword by Victor F. Marshall very properly states that this is a full account of available diagnostic methods. However, in stating the four stumbling blocks to truth, he, himself, falls into error's way by quoting the statements as being those of Roger Bacon, whereas in point of fact, they are those of Francis Bacon. With this small proviso, this book is heartily recommended.

ATLAS OF HAND SURGERY. Marc Iselin and his associates, Luc Gosse, Serge Boussard and Daniel Benoist. Translated by John C. Colwill. 325 pp. Illust. McGraw-Hill Company of Canada Limited, Toronto, 1964. $21.15.

The author's declared intention is to provide an atlas of surgical techniques to complement his earlier text, "Chirurgie de la Main", in which principles of hand surgery are presented. This atlas has been well translated.

Basic chapters on instrumentation, anatomy, physiology and splinting are included in the first part. However, the initial management of the injured hand, basic techniques of dressings and the treatment of the acute burn are not included. The remainder of the book covers many of the operative procedures used in hand surgery, and is well illustrated by nearly 700 line drawings, but no photographic illustrations.

To those familiar with other textbooks on hand surgery, this provides an interesting comparative study of techniques. However, the techniques described are clearly the personal choice of the author and many methods currently favoured in other centres are not mentioned. The section on congenital abnormalities covers only the more simple conditions, and there is no section on tumours of the hand or on rheumatoid arthritis.

This book is interesting in concept but it is regrettable that it does not offer a wider and more complete coverage of the techniques employed in hand surgery.

LA RESECTION-ANGULATION DE LA HANCHE EN DEUX TEMPS. René Charry. 540 pp. Illust. Editions Doin, Deren & Cie, Paris, 1964. 145F. $29.00 (approx.).

Il s'agit d'un volume de 500 pages qui décrit d'une façon un peu romancée et dramatique par moments, non seulement une technique opératoire et les résultats obtenus, mais aussi l'histoire d'une intervention chirurgicale, depuis sa conception par l'auteur, ses difficultés de la faire accepter par les confrères, sa réalisation et les résultats obtenus, les témoignages d'orthopédistes d'Europe et des Etats-Unis.

Le livre, qui veut prouver la valeur de la résection-angulation de la hanche en deux temps pratiquée à 15 jours d'intervalle, est très abondamment illustré de photos, de clichés et de dessins.

La technique opératoire est rigoureuse, très bien décrite et jusque dans ses moindres détails.

Suit un chapitre relatant les opinions des orthopédistes sur cette intervention et un chapitre où l'auteur s'applique à répondre aux problèmes posés par l'intervention; ses variantes, ses modalités, ses indications etc.

Dans ce chapitre et les pages qui suivent, l'auteur réitère ses opinions, les explique et les défend.

Les indications opératoires sont aussi bien précisées. Et l'auteur termine par une analyse détaillée des résultats obtenus chez les patients qu'il a opérés.

En somme, c'est un livre bien documenté, qui défend une idée, avance une opinion, et nous la fait partager dans un style agréable et très convaincant.

ON THE NATURE OF NEOPLASIA IN MAN. D. W. Smithers. 176 pp. Illust. E. & S. Livingstone Ltd., Edinburgh; The Macmillan Company of Canada Limited, Toronto, 1964. $4.50.

Dr. Smithers has brought together in an interesting little book, eight of his recent lectures. His main purpose is to call into account the conventional theories about the nature of neoplasia in man. He is critical of the 10 following characteristics currently attributed to cancer: that (1) cancer is a specific disease of cells; (2) a cancer cell is one that has been permanently changed and is no longer capable of behaving like a normal cell; (3) cancer cells multiply without restraint; (4) cancer cells grow at the expense of normal tissues, actively invading and destroying them; (5) cancer cells can gain access to cavities and lymphatics and are capable of developing into new tumours wherever they come to rest; (6) if the cause of cancer in a cancer cell could be discovered the whole problem would be solved; (7) every cancer cell must be removed or destroyed *in situ* if the patient is to be cured; (8) if one viable cancer cell remains, treatment will fail; (9) a chemical poison specific for cancer cells may one day be found to replace all present treatment methods, and (10) that when the disease has spread beyond the scope of local removal or irradiation, any non-specific cell poison may be worth trying. He marshals many years of clinical experience to point out the incompatibilities of these postulates, namely, many cancers are multicentric in origin and have a long prediagnostic natural history. The growth of cancer is not a straight-line progression, but has variation in speed and spontaneous regression that may be observed without treatment. Many cancers have been shown to be hormone dependent, thus invalidating the proposition that they are entirely autonomous and not under the growth-regulating mechanisms of the body.

Smithers postulates that the behaviour of the individual cells is in fact a result of and not the cause of disorganized growth regulation within the tissue. The soil may be as important if not more so than the seed in establishing the growth patterns of cancer. He has vigorously challenged the basic scientists to broaden the scope of their research and not to restrict their studies to single cancer cells taken out of their natural environment but to study the problem as a complex phenomenon of many interdependent cells.

This book is a stimulating one to those interested in cancer; it will be of particular interest to clinicians and provocative to the basic scientists.

YEAR BOOK OF CARDIOVASCULAR AND RENAL DISEASES. 1963-1964 Series. Edited by W. P. Harvey et al. 541 pp. Illust. Year Book Medical Publishers, Inc., Chicago, Ill., 1964. $11.00.

This is the third of the new series of Year Books to be published; the series began in September 1963. The book is divided into the following sections: normal and altered cardiovascular function; cardiovascular disease in infants and children; heart disease in adults; the coronary arteries and coronary heart disease; hypertension; pulmonary circulation; vascular disease; cardiovascular surgery; renal disease, and techniques.

As is usual with year books, this one abstracts the best articles of relevant topics for the previous year. The obvious and only fault with such a book is that the articles are usually many months old by the time they are published in journals, and the year book itself is necessarily much more behind when it is finally published. However, in spite of this, the year books make an extremely valuable set of reference books for general use.

The comments of the special editors are extremely valuable, because they add perspective to individual articles, the authors of which may not be well known. A single reviewer could not be familiar with all of the editors of this year book but if they are of the eminence of John Kirklin, the book must be highly recommended.

PATHOPHYSIOLOGY OF PEPTIC ULCER. Edited by Stanley C. Skoryna. Foreword by Henry L. Bockus. 497 pp. Illust. McGill University Press, Montreal, 1963. $20.00.

This book consists of the collected papers from the Second World Congress of Gastroenterology held in Munich, Germany, in 1962. Most of the authors are scientists and clinicians who are well recognized for basic and original research on digestion.

Although each individual paper is complete and valuable in itself, they have been grouped under five different headings and they leave very few gaps in the coverage of the subject matter. Basic physiology and biochemistry, experimental ulcer production, the effects of pharmacological agents and local and systemic factors are considered in a very comprehensive fashion. Although some papers may appear too technical for a clinical surgeon – this is unavoidable when dealing with membrane potentials, electron microscopy and basic biology — each one is followed by an excellent summary which is invaluable to the average reader.

The last chapter of the book was written by Dr. Skoryna and deals with "Stochastic Processes in the Causation of Peptic Ulcer". His is an ingenious attempt at constructing a mathematical model of the pathogenesis of peptic ulcer. It appeals to the reader's imagination in a challenging fashion by linking this particular problem with the limitless world of philosophy.

The book is well printed on excellent paper and nicely bound. The illustrations are of the highest quality and the tables highly comprehensive.

Dr. Skoryna's book is a wonderful collection of reference material which provides up-to-date information. It is not a handbook to be used in daily practice, but it will help the inquisitive surgeon in his study of peptic ulceration.

THE LAWS OF BONE STRUCTURE. H. M. Frost. 167 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1964. $9.00.

This monograph deals with some important physiological mechanisms and laws which regulate growing, moulding and remodelling of living bones. It is concerned with behaviour of bone cells in response to physical deformation of bone. The author proposes that the size, location and structure of bones are largely determined and regulated by a physiological mechanism which he calls "space polarization of bone cell activity", that is, the orientation of bone cell metabolism, division and movement.

He describes the basic mechanism as follows: Deformations of bones caused by physical loads generate "signals" at the deformed bone surface. These signals selectively activate and inhibit particular bone cells. For example, osteoclasts are activated and osteoblasts are inhibited at the convex side of an angulation deformity to remove bone, and *vice versa* at the concave side to add bone — a process which he calls "drift" of bone. The signals are then automatically reduced by the cellular activities — a negative feedback mechanism, and the bone deformation is corrected.

The author states that this mechanism of space polarization of bone cell activity operates consistently and thus the outcome in the bone structure is consistent, reproducible and predictable. On this basis he formulates these properties of bone into laws of bone structure.

It is surprising that this book on the laws of bone structure fails to mention Wolff's law, when in essence the author is describing the mechanism of Wolff's law. The law states that changes in the internal architecture and external form of bone follow the changes in physical forces which act on bone in accordance with mathematical laws.

The book is rather difficult to read and understand because many new and specialized technical terms are used (although they are defined in a glossary) and many complex mathematical terms and formulas with more than 80 symbols are used. It also fails to furnish experimental evidence to support the author's ideas.

The book contains new and important ideas and it should interest scientifically minded orthopedic surgeons and some physiologists, biophysicists and biologists who are interested in bone and cellular behaviour to physical stimuli.

THE OBSTRUCTING ACROMION. Underlying Diseases, Clinical Development, and Surgery. Bernard Diamond. 212 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1964. $11.50.

This book of 212 pages is published by an author who has had over 18 years' experience in the practice of orthopedic surgery. It is well written and easy to read. It consists principally of a series of case reports, all of which are well documented and beautifully illustrated and are accompanied by radiographs and photographs. The book describes the anatomy, physiology and pathological conditions of the shoulder joint as related to the acromion. The author has had the opportunity of performing 44 acromionectomies, either in part or *in toto*. In Chapter 2 he describes a new method for the excision of the acromion and also demonstrates a "shoulder board" for positioning the patient during this operation.

The book is worth reading and emphasizes the part played by the acromion and the acromioclavicular joint in pathological lesions of the shoulder joint.

CORRECTABLE RENAL HYPERTENSION. Chester C. Winter. 190 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1964. $8.25.

This book is a summarized account of the current state of knowledge regarding the kidneys and hypertension. It makes no claim to being comprehensive. The author is at his best in describing methods of diagnosis in cases of renal hypertension, both for screening and for more intensive study. Particularly well covered are retrograde renal studies and tests making use of radioisotopes. The surgical aspect is briefly covered. The section dealing with prognosis is interesting, but, as the author himself indicates, it is probably too comprehensive for most centres. Altogether, the book is an adequate summation of the present trends in renal hypertension and newcomers to the field especially should find it of value. There is an extensive bibliography.

SURGICAL CLINICOPATHOLOGICAL CONFERENCES OF THE MASSACHUSETTS GENERAL HOSPITAL. Edited by Benjamin Castleman and John F. Burke. 249 pp. Illust. Little, Brown and Company, Boston; J. P. Lippincott Company of Canada Ltd., Montreal, 1964. $13.50.

This 249-page volume contains 50 selected surgical cases presented at clinicopathological conferences (CPC) of the Massachusetts General Hospital and published in the *New England Journal of Medicine*.

The history and development of the CPC as a teaching method are outlined.

The cases chosen were discussed during the past 25 years, the majority of them being within the last 10 years. In the light of modern diagnostic refinements, earlier cases have been excluded since they are proportionately less instructive.

A wide range of clinical surgical problems is encompassed by the 50 cases which are presented by a group of distinguished discussants, including Reginald H. Smithwick on shifting colicky abdominal pain, Robert R. Linton on a patient with a mass in the region of the ileocecal valve, Michael E. DeBakey on chronic phlebitis, abdominal mass and multiple pulmonary densities and Robert M. Zollinger on recurrent bleeding uncontrolled by gastrectomy.

Many of the surgical cases conclude with the operative findings, in contradistinction to medical CPC's which usually reach the postmortem stage. In these cases additional follow-up history has been provided as addenda to the presentations.

In those instances where recent advances in knowledge of the condition under discussion have been made, comments by the original discussant, the pathologist or some other eminent authority render the presentation more timely.

The excellent clinical discussions are enhanced by abundant reproductions of roentgenograms, gross specimens and photomicrographs. The table of contents, list of discussants and the index enable the reader to single out a case on the basis of discussant, clinical features and anatomical diagnosis.

This collection of surgical CPC's is recommended to the reader as a rich source of clinical information as well as for the pleasure that can be derived from matching diagnoses with those of outstanding experts.

ACTION OSTEOGENETIQUE DE LA GREFFE DE MUQUEUSE VESICALE. R. Dozinel. 118 pp. Illust. Editions Arscia S.A., Brussels, 1964. Fr. B. 220. $4.40 (approx.).

Le but de l'auteur est d'étudier l'origine de la cellule osseuse en se servant de l'ossification hétérotopique au moyen de la greffe de muqueuse vésicale.

De nombreux expérimentateurs, dont Huggins, ont depuis longtemps établi le pouvoir ossifiant de la greffe de muqueuse vésicale. Deux hypothèses ont été proposées pour expliquer cette ostéogénèse.

La première théorie, la théorie cellulaire, soutient que l'ostéocite se forme à partir du greffon. La deuxième théorie, la théorie d'induction, soutient que l'os provient de la transformation des fibrosites de l'hôte en ostéocite sous l'influence de la greffe.

L'auteur a procédé à trois séries d'expériences en greffant de la muqueuse vésicale autologue, homologue et hétérologue, dans la chambre antérieure de l'œil, dans le muscle strié et dans le rein de 950 cobayes. L'auteur tente de démontrer successivement que les ostéocites ne proviennent ni du greffon, ni du tissu conjonctif autour de celui-ci. Il rejette donc les deux théories de l'ostéogénèse hétérotopique.

Il reprend alors une troisième théorie, la théorie hématogène, qui soutient qu'il y a dans le sang circulant des cellules de potentialité ostéogénique. Il explique alors comment cette théorie est bien vérifiée par ses expériences personnelles. Il ajoute, cependant, un nouvel aspect dans cette théorie, c'est l'ostéotaxine.

En effet, l'action conjuguée de la muqueuse vésicale et du tissu conjonctif autour de la greffe serait le facteur indispensable à la diapédèse des cellules à potentialité ostéogène au niveau du foyer de la greffe.

En conclusion, nous pouvons dire que ce volume est intéressant, que les expériences ont été bien menées, que le texte est étoffé de plusieurs références et que l'originalité des hypothèses émises par l'auteur marque un pas en avant dans la compréhension du problème complexe qu'est l'ossification hétérotopique. C'est un livre qui sera lu avec avantage par tous ceux qui s'intéressent à ce problème.

THE CARE OF THE INJURED. P. A. Ring. 164 pp. Illust. E. & S. Livingstone Ltd., Edinburgh; The Macmillan Company of Canada Limited, Toronto, 1964. $4.50.

Unfortunately, the reviewer takes exception to the author's preliminary remarks where he states "the responsibility for the organization of accident services . . . must properly be regarded as the responsibility of orthopedic units".

It is difficult to determine to what level of training this small book on traumatic surgery is aimed. The most basic and elementary features of the care of injuries are presented in abbreviated form in orderly fashion with, of course, the concentration of the book being on fracture work and other orthopedic problems.

The reviewer feels that the book can be best described as a good introductory book to other introductory books on emergency surgery which have long been established and accepted in the teaching of surgery.

TROPICAL SPLENOMEGALY. A. K. Basu and B. K. Aikat. 195 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1963. $11.25.

This short book is a clinical study of 190 patients with enlarged spleens seen in Calcutta where this entity is commonly labelled 'tropical splenomegaly'. The clinical and pathological findings are given in considerable detail and references to the literature are supplied concerning portal hypertension, cirrhosis of the liver and hypersplenism. Of 97 patients operated upon (and reviewed in more detail), 67 had primary liver disease, and, of these, 50 had post-necrotic cirrhosis. Extrahepatic obstruction was noted in 15 patients, and in the remaining 15, specific diseases of the liver were encountered including hemoglobin-E thalassemia—13, kala azar—1, malaria—1.

The authors conclude that "the majority of the cases of so-called 'tropical splenomegaly' result from hepatic pathology. A smaller number manifesting the same clinical syndrome is due to extrahepatic obstruction and to other morphological variants of hepatic injury. A separate clinicopathological entity of 'tropical splenomegaly' peculiar to this geographical area of India need not, therefore, be recognized."

They also describe experiments with dogs which seem to indicate that the spleen exerts an inhibitory effect on the regenerating capacity of the liver. They commented that "In cases of macronodular cirrhosis, there is sufficient clinicopathological and experimental evidence to justify the performance of splenectomy with appropriate shunt procedures as an effective therapeutic measure."

This book contains a wealth of material for those interested in the problems of portal hypertension and splenomegaly.

OBSTETRICS. J. M. Holmes. 256 pp. Illust. Baillière, Tindall and Cox Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1964. $3.00.

Since 1876, Messrs. Baillière, Tindall and Cox have been producing "concise medical textbooks" suited to the pocket and the pocketbook of the medical student who chooses to use them. Continuing with this tradition, the author has made an ambitious attempt to compress nearly every topic from pelvic anatomy to craniotomy into 256 small pages. Unfortunately, present-day obstetrics is not a subject which can be condensed. In recent years, enormous advances have been made in our basic understanding of the endocrinology of reproduction and of the physiology of the uterus, placenta and fetus. A meaningful summary of these important areas together with their far-reaching clinical implications is all but impossible in a book of this size. If a discussion of the endocrinology of the menstrual cycle is to be included, it cannot be dismissed (as it has been) in 1¾ pages. It is, for instance, by no means settled that the granulosa cells of the maturing follicle secrete any of the estrogens, let alone estradiol.

Furthermore, no amount of description will replace an adequate number of high-quality illustrations in the presentation of, say, the mechanism of labour. The cost of such illustrations precludes the production of an inexpensive textbook.

This book lacks the sense of proportion so essential to one of its size. There is, for instance, far too much space given to the incarceration of the retroverted gravid uterus, an unusual clinical condition.

There is a tendency to over-classification and this has led the author to include toxemia of pregnancy under "displacements of the gravid uterus" and antepartum hemorrhage under "extrauterine gestation".

One is alarmed to read that "if the fetal head passes through the brim of the pelvis, safe vaginal delivery is usually possible" and that "first labour with an unscarred uterus is the ideal trial of labour". It is to be hoped that the broader view in 1965 would include the fetus in its consideration as well as the mother.

Several obstetrical practices are referred to which are peculiar to the United Kingdom and which are not followed in North America.

One hopes that obstetrics will appeal to students as something more than a set of well-delineated clinical situations described by a glossary of antiquated terms and managed in a rigidly empirical fashion. "Canned" obstetrics is not the answer to the student's dilemma. The reviewer cannot honestly recommend this book for use by medical students.

(Continued on page 238)

(Continued from page 236)

CARDIOVASCULAR SURGERY 1963. American Heart Association Monograph No. 7. Council on Cardiovascular Surgery, American Heart Association Scientific Sessions, Los Angeles, Calif., October 25-27, 1963. Edited by F. A. Simeone. 180 pp. Illust. The American Heart Association, Inc., New York, 1964. $3.00.

This supplement to *Circulation* contains a carefully selected group of 30 papers presented at the Annual Scientific Sessions of the American Heart Association. A wide range of topics on experience in congenital, acquired and experimental procedures has been chosen.

Six articles deal with prosthetic valve replacement. The physiological changes experienced during prolonged by-pass are discussed, as well as the influence of dilation techniques, hypothermia and mannitol. Transplantation of the heart and hyperbaric oxygen are considered, and there are interesting papers on artificial hearts and self-energized pacemakers.

For those interested in the surgery of congenital defects, there are reviews of experience with pulmonary-artery banding, transposition and subaortic stenosis. Arterial surgery for arteriosclerosis of small vessels is also discussed.

This review is extremely interesting, and is to be recommended for those interested in the field and as an addition to every hospital library.